CAUSES OF BLINDNESS IN AND AROUND SURABAJA EAST JAVA, - INDONESIA

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Thesis by Johanna ten Doesschate,
born 2 May 1912
at Steenderen, Holland.

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PROMOTOR : PROF. DR. I. SALIM

CAUSES OF BLINDNESS IN AND AROUND SURABAJA, EAST JAVA, INDONESIA.

TESIS

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OLEH

JOHANNA ten DOESSCHATE

dilahirkan di Steenderen

A thesis submitted to the Medical Faculty of the University of Indonesia in Jacarta in partial fulfillment of the Requirements for the Degree of Doctor of Philosophy.

Djakarta, Indonesia
1968

Aan de nagedachtenis van
mijn Ouders.
Dan dipersembahkan kepada
Farida dan Ferial Basoeki.

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At the end I cordially thank the printers, N.V. Rahman Tamin, for the understanding way they have complied to my wishes.

N.B. The order of the Chapters is irregular : Chapter 9, 11 & 12 are printed at the end, after Chapter 30.

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General Introduction.

Chapter one.

Everybody who has worked in an eye hospital in East Java, from Steiner (1894.480) who made the first survey among the blind of Surabaja in the nineties of the last century, until the present time, has been struck by the large number of blind persons seen, which is much higher than the number seen in a comparable institution in Western Europe.

Part of the blind patients have been blind for a long time, part of them only recently suffered a blinding disease. Unfortunately only a small part of them can be helped, another group remains irreparably blind. From this fact alone it is already clear how important preventive measures would be.

Apart form the impression of a large number, it is rather difficult to estimate how many blind people there really are in the region, because the last census of the whole population, which counted the blind separately, has been held in 1930. There is a survey now going on, made by the Djawatan Sosial (Social Service) in an attempt to ascertain the incidence of blindness. Unfortunately the figures for East Java Province are not yet complete : only half of the ,,Kabupatens" (former Regencies) have completed their returns, using laymen as investigators and finger-counting at 1 metre in front of the eyes as visual limit. Mr. Soejoso from the provincial Social Service was kind enough to allow me to use the figure which has as yet been found, i.e. ca.6.500 blind persons, in nearly half of the province. This figure is to be taken with all kinds of reservations, but it is more or less in accordance with the figures which have already been collected from the other provinces of Java (personal communication of Mr. A. Moh. Arief, director of Rehabilitation of the Sosial department of the central government). Part of the reservations with which these figures have to be taken is, that even in advanced countries it is difficult to catch really all blind persons. Mrs. Schappert-Kimmijser (1959. 414) who conducted a survey by questionnaire in the Netherlands, states that either the blind themselves or their family are often reluctant to admit the fact. This is all the more true in this country, where various kinds of superstition are yet active, which look for the "guilt" in cases of blindness either in the blind themselves or in their parents or relatives. For that reason especially blind children sometimes are hidden for fear of comment by the neighbors, which can happen in distant regions, and which already has been mentioned by Tijssen (GTvNI LXXVI, '36, p. 2895).

Another reservation in considering the figures of the Social Service survey is that they are collected by laymen, and are not checked by experts. It is uncertain if the visual limit is really adhered to, and in the same degree, by all investigators.

In most of what is wrttein on the incidence of blindness in this country recourse is still had to the figures of the 1930 census. They are multiplied by the population increase in a rather impressive way, because the population has doubled in the intervening years. The census used the same limit of visual acuity as the present survey of the Social Service, and stated for Java and Madura: more than 100 000 blind persons, to which had to be added more than 30 000 for the other islands. Mulock Houwer in his "Rector's Address" 1940 (311) estimated that if the census figure would be "corrected" to make it more comparable with European/American figures, there would be 668 000 blind people (taking less than 1/30 as the limit of visual acuity) in Java and Madura. This figure has been used also by Oey Khoen Lian (1953.333) and prof. Salim (1959.406) as a startingpoint. For the year from Aug. 1st '66 - Aug. 1st '67 I compared the figures for the number of blind people, using the limit of less than 1.5/60 and that of less than 1/10, and I found not nearly a factor of 5 like Mulock Houwer supposed, but for children only 25% had to be added to the number of blind with the lower limit, and for adults : 78% for men and 84% for women. This would lower the estimates for the total of blind people, even if the higher limit of visual acuity were used, very considerably. Probably the true figure lies somewhere between the present very low figure of the Social Service survey and the former estimates.

If we take the census figure of 1930 as certain, multiplying it by 2 for the population increase, we would get 272 000 for the whole of Indonesia; as East Java has ca. 20% of the whole population inside its borders, there would now be more than 50 000 blind persons in East Java Province. If we see that in a period of 5 years almost 5 000 blind patients came to be treated in one Eye Hospital only, this figure does not seem improbable. It would give a rate of 250 : 100 000 inhabitants. This is comparable to other tropical countries. It is admitted that this is a conservative estimate, but still it is a high percentage, and 5 times as high as that in Japan or the Netherlands.

The investigation into the causes of the several thousand blind persons who were examined at the Undaan Eye Hospital has not the value which an overall survey of the whole population would have, but it can give useful data on the relative importance of the causes of blindness in this area. The results cannot be generalized for the whole of Indonesia, because it is quite certain, that the various regions of this large and varied archipel are not affected by the same eye diseases in the same relative frequencies. A striking example of this fact can be found in the Reports which the Djawatan Kesehatan Mata (Government service for Eye Health) has put together (336. 337. 338) ; whereas in East Java conjunctivitis gonorrhoica ranks high as an eye disease of all ages and especially as a blinding disease, there are certain regions as e.g. Maäs points out also (1960. 250) where it is very rarely seen, if at all. It has long been known too, that Trachoma is unevenly distributed, possibly following traffic, as was seen by Warouw in 1935 (1935. 548) and others before and after him.

Consequences of the high blindness figure.

Especially in the economic and social conditions as they are here and now it is not necessary to dwell long on the amount of suffering which the high number of blind persons have to carry : the burden to themselves and to their families seems even heavier than formerly. In the former stable society the blind in the village could get their food from the large family, and sit somewhere in the shadow. In the developing society at present, firstly it is economically much more difficult to have members of the family who do not take part in the production process, and also the thought that blind people CAN work if properly trained is seeping through. Also hope that modern medicine can maybe cure blindness is spread more widely than it used to be. The suffering which all of this entails for the blind themselves and for their families has increased. Also the realization that the economy as a whole cannot go on carrying the extra cost of many invalid members of the community makes for enhancing the importance of a search into the causes of blindness and eventually its prevention.

As the situation stands now a very large part of the blind are not taking apart in the production process. There are a few Training Cenrtres for the Blind, and there are some blind people who have got a higher education : these few are all the more to be admired in that they in the face of tremendous difficulties have managed to learn a job or a profession, in which they do valuable work, making them real pioneers, who blaze a trail for more blind people to follow. But the problem as a whole does not yet receive enough attention, especially as a problem in preventive medicine.

If it would be certain (as can be guessed) that a high number of the blinding diseases in the country is preventable, then it would become **morally necessary** to devise measures to prevent them as soon and as effectively as possible. This is important for all blinding diseases, but especially so for those diseases which strike at a very low age, in babies and children, because if they survive, they have many blind years to live, a personal burden and an economic burden for the community. The difference between a child of say two years of age who becomes blind, and has to be educated on this basis, and a person of 50 yrs of age, who goes blind maybe by a slowly progressive disease, which allows him to adapt himself to the situation, is enormous, not only from a personal view, but also in its further ramifications, like the special educational service which will be required first and the special facilities to find him a job afterwards. The child which is going blind at a very early age (like hundreds of them do in this area) is comparable to a child born blind. Even with the best educational care these people will always miss the whole human experience which is based on eyesight ; whereas people who go blind later in life have developed their personality on the experience gained by their eyes, which makes them emotionally more vulnerable, but otherwise they can be trained for more diversified kinds of work.

The first step in the **prevention** of any kind of disease is to get a clear pictures of the **cause** or causes of the disease. Blindness is not a disease, but a complex results of many kinds of eye diseases which differ from climate to climate, from country to country, from region to region and also vary according to age, sex and kinds of work. Therefore any kind of generalization is impossible: for every distinct region the complex picture of diseases which produce blindness at the various ages of either men or women has to be studied for itself.

Other countries.

Until now only Great Britain can give an almost comprehensive account of the number of blind people and of the cause of their blindness, as for many years already there has been legislation which makes it possible for the blind to get compensation, if they register as blind. Formerly certificates for blindness could be issued by general medical practitioners, but now these cases are more and more seen by competent oculists. Sorsby (470. 471) has published several reports on the data gathered in this registration, but even he states (472. 1966) that the registration does NOT comprehend all cases.

In a few States of the USA where legislation made this possible there also have been published reports on the prevailing causes of blindness, but in most of the other States of the USA they can only be estimated (Sight Saving Review. 184).

In the Netherlands Mrs. Schappert-Kimmijser has used questionaires sent to all oculists, schools for the blind, and all the various organizations involved in work for the blind, resulting in data covering more than 80% of all blind persons (414)

Form the Eye Clinic at Basle, Switzerland, there have been published figures for the causes of blindness seen in patients during a certain period (Matiaska 275, Willome 576), which investigations are more or less on the same lines as this present study as they report on patients seen in a central eye hospital.

Recently WHO (1966) published a Survey on Incidence and Causes of blindness (568) which contains the data published to date all over the world. Large areas appear not yet to be covered in this survey, and the list of definitions which are used in various reports for defining blindness show 65 numbers. Therefrom it is clear that it is not yet the last word on the subject, many more will follow before there is a really complete picture of the problem, but for the time being it gives valuable information, all the more so as it points out gaps in our present knowledge.

Indonesia.

In recent times Oey Khoen Lian has given figures on the incidence and causes of blindness for North Mid Java (1953. 333), Mrs. Seniman (= Go-Sennema) published a survey from the Bandung Eye Hospital (1963. 150), Maas investigated the problem for North Sumatra (1960. 250). For East Java there exist the old figures of Steiner (1894. 480) from 70 years ago.

The very densely populated province of East Java (Size ca. the same as the Netherlands, population almost double as large!) was as yet not represented in recent lists. The figures used by Prof. M. Soetopo at the Bandung Seminar on Blindness in 1964 were taken from the present study.

In April 1968 there appeared data on the years 1962 & 1963 from the Eye Dept. of the Medical Faculty at Surabaja (Nj. dr. Tamin & Ngurah Oka 1966. 498).

The only investigation which was practicable under the circumstances was to count the patients of a large centrally situated eye hospital and register the blind among them. Rumah Sakit Mata Undaan at Surabaja is a wellknown hospital which has continuously been in business for ever 50 years now, and which contains 50% of all beds for eye patients in the whole of East Java Province. The other larger eye centres are the University Eye Department at Surabaja, which sees as many outpatients anually as the Undaan hospital, but which has only half as many beds and the Eye Department of the general hospital at Malang, which sees half as many outpatients and has still fewer beds. In the whole of the region East Java and Madura at the time of the present study there were active ca. 7 qualified oculists (2 of them at Undaan Eye hospital).

Chapter two.

Type of investigation.

One could imagine several types of investigation into the number and causes of blindness in a region : it would be possible to select a small part of the region, maybe a ketjamatan (district) and collect all the inhabitants, count the blind and try to ascertain the cause of their blindness. This result could then be generalized for the whole region. The drawbacks would be many : if house to house visiting is not done it is not certain that one would see all blind persons. If nothing therepeutically is done at the same time the investigation would not be welcomed. This type of investigation could profitably be combined with other types of public health action, e.g. mass vaccinations. But even so the results would not be permissible for generalization if there would not be test areas in all kinds of terrain : coastal plains, mountain villages, great city, small towns etc. Also the kind of food would have to be taken into account : is there fish, is rice the staple food, or maize or cassave.

Another type of investigation could be conducted by sending questionnaires to the village heads, who generally know their people quite well. Data on the number of blind persons and their approximate age could be collected in this way, but as to the causes one would remain in the dark, and quite possibly the returns would not come in a usable form, or even not at all (we tried this method in Surabaja it self, where not more than 10% of the heads replied at all).

In several other countries there have been published figures based on the inmates of schools or other institutions for the blind ; there is only one school for the blind and one workshop in the whole of the province East Java, which makes this method impracticable.

In connection with the planned compulsory education for all children of school age there were certain "Trial Areas" where this was tried out. By an initial registration all the children were caught and a note was made if they by one handicap or another would be unable to follow a normal school. In practice this registration after a few years already proved unreliable, as people had moved, or only moved the children to other relatives, which makes the registration unusable for an investigation into incidence and causes of blindness.

The British type of registration of the blind based on social welfare measures for them is still out of the question in this country, where there is as yet no legislation for the handicapped (like e.g. the Philippines have had for several years).

Present type of investigation.

Trying to make the best of it, we were reduced to just collect the data from the patients who came to the Outpatients' Department and who were or became blind, and to analyze them as far as possible. In many cases the diagnosis could be made under much better circumstances than one would have had in a field survey, because many of the patients were followed over a period of time, were admitted, were seen by other specialists in consultation etc. If they were seen for any length of time, they were always cared for by the same qualified oculist, which decreased the possibility for misunderstandings of other people's notes, but because we were two, we could consult one another on difficult cases.

If the number of patients seen in this type of investigation is large enough, one can assume that they give a fair sample of the blind persons in a region. From the relative importance of the blinding diseases seen among these patients conclusions can be drawn as to the importance of these diseases in the whole region. During the period under study we saw nearly 1.000 blind people annually, which makes the sample rather large. Of course there are many more blind people who never come to an oculist : lack of initiative, fatalism, lack of money or lack of a guide, are usual reasons.

Information about the facilities for medical care of eye patients should be far more widespread, and village heads and other influential persons should try to send their dependents for **early** examination. All types of information services (djawatan penerangan) could find a large and profitable field of work in spreading information sbout the public health aspects of eye care, but they will have to be informed themselves first.

Chapter three.

Definition of blindness.

In international ophthalmological circles there have been long and complicated discussions through the years about a generally acceptable definition of blindness. Not only oculists, but also legislative bodies have been implied, the latter in countries were there is a system of compensation either for the blind alone or also for other types of handicapped people. In those countries naturally a legal definition of blindness is required which does or does not take into account the visual acuity, the size of the field of vision, and possibly the remaining near vision.

The discussion centered not on the definition of total blindness which is generally accepted as visual acuity = 0 in both eyes, but on the definitions of concepts like "social blindness", "Pedagogical blindness", "industrial blindness", "high weaksightedness", "weaksightedness" e tutti quanti. Not even all experts in the same country are agreed on the boundaries between these several degrees of blindness. All of this is important in countries where there are different types of schools for various degrees of weak-sighted or blind children, and where there are social services helping the blind in job placement, in additional training, or in getting a pension. In consequence many non-medical officials are involved in the work, and they must be able to rely on a definition of blindness in its various degrees to be able to implement the law. The eye doctor determines the degree of blindness and after that the social worker steps in and tries to find the best way of helping the blind within the frame of the law. In general it can be said that in highly industrialized countries people with bad eyesight cannot be placed in the ordinary production process, but on the other hand large factories are often willing to organize special facilities to give work to handicapped people, especially in times of shortage of labor. The decisive condition is that the blind persons must be **educated well enough** to be able to fill a job : the most modern job for which blind people have been succesfully trained is that of programmer for computers, and there are many blind typists and telephone operators, who can earn their living as well as seeing persons. In this country which is still an agricultural one, many nearly blind people can still be employed in their old jobs, or helping others, but in this field also it will be necessary to prepare for industrialization, i.a. by educating the blind.

The definition for economic blindness used in the "Blind Persons Act" of the United Kingdom (1920) is : "To be so blind as to be unable to perform any work for which eyesight is essential".

According to Sorsby (1966. 472) the practical limit applied is 3/60 or less.

The Netherlands Association for the Prevention of Blindness uses :

1. Absolute blindness : no light perception at all.
2. Social blindness :
   A. Inability to take part in normal traffic by insufficient sight, without the use of special aids.
   B. Inability to do work normally done by sighted persons without the use of special aids.

In ophthalmological practice this means: visual acuity of maximal 2/60.

Visual field not larger than 10 degrees.
In Switzerland Matiaska and Willome (Basle) used these limits :

1. Social blindness : 6/60 — 1/60.
2. Practical blindness : 1/60 — correct light projection.
3. Absolute blindness : Faulty light projection — visual acuity = 0. (275. 576)

Budden in Nigeria (1956. 45) worked out a definition of blindness by observing people with impaired vision and the work they were still able to do, and found that ability to count fingers up to and including at distance 3 Metres (3/60) embraces in Nigeria practically all persons NOT working, or engaged in blind occupations, as a result of loss of vision.

For the purpose of deciding on a just recompensation for a handicap in highly industrialized countries there are several other definitions in use :

1. Economic blindness, making a man unable to do normal average work.
2. Occupational blindness, which makes a man unable to follow normal (former) work.
3. Educational blindness, preventing a child from attending normal school classes.
4. Social blindness, which includes all people who are markedly handicapped in the fulfilment of the obligations of normal daily life.

These various definitions are needed in countries where social welfare legislation provides compensation of handicaps suffered during work and where revalidation is organized on a legal footing. In Indonesia one single and simple definition for blindness is sufficient for the time being.

The search for a definition of blindness which would fit the local circumstances and which would also make my investigations comparable with those in other regions, could end when I heard that there was an International Standard Classification in the making, which was designed to end difficulties and uncertainties. The definition prescribed by the International Standard Classification leaves the investigator free to choose his own borderline between blind and seeing, so as to get the highest possible usefulness from the data collected.

It is needless to say, that not all requirements could be fulfilled to make our data perfect for all purposes. Even those data could often not be completed perfectly : fields of vision are difficult to plot if patients do not understand the meaning of fixation, all the more so if their vision is weak.

In accordance with former usage in this country and the needs as they exist to day and here I chose as the limit of visual acuity $1^1/_2/60$, i.e. below that limit I called a person blind. This is the same limit which was accepted by the Seminar on Blindness at Bandung in 1964 (518) and which has been used by other investigators (Oey, Seniman, Maäs). It is also the same which was used in the census 1930, and which is now being applied by the investigators of the survey for the Social Service. It is easy to apply, to explain to lay workers, and it delimits in the Indonesian context at present the people who are "really blind", i.e. not able to perform any gainful work which normally is done by sighted persons.

Chapter four.

Descriptions of the patient material.

All patients, who came to the Outpatients' Department of Undaan Eye Hospital at Surabaja during the years 1958 through 1962 and who were finally blinded, were counted and classified. Patients in private practice were not considered. A general survey of the whole area under investigation would of course have given a more accurate picture of the total number of blind people, but it was felt that the large number of blind persons seen in five years' time, gave a sufficiently varied and fair impression of the main causes of blindness and of their relative importance. The one persons who became blind through snakebite as opposed to the many who became blind by syphilis, leads to the correct conclusion that in this area syphilis is more dangerous for eyesight than snakes.

Social status.

The blind persons classified as blind in this investigation, are to be considered as "unselected material seen in an eye clinic". The Eye Hospital Undaan is meant for help to the unprivileged classes and charges only a very low fee or none at all, according to the circumstances of the patient. In this country this means that most of the patients were native born Indonesians, from Javanese and Madurese stock, whereas there comes only a small number of persons of Chinese and Arab extraction to an outpatients' department. The work done by the patients comprised : unskilled labor, small vendors, servants, peasants, artisans, also military and police. It is probable that there is not so much blindness in the more well-to-do groups of the population, as part of the blinding diseases is certainly connected with poverty, dirt and ignorance. It is estimated that our patients belong to the 80% of the populace who are poor. It can be taken as more or less representative of the whole population.

Place of origin.

We thought at one time, that it would be feasible, to divide the patients in "rural" and "city dwellers". But it became clear that a not unimportant part of the population in the area is semiurbanized : they go back and forth to their village of origin, to help at harvest time (and receive their wages in natura) and to celebrate special events in the family. Families are often not living together permanently ; Islamic law allows a husband up to 4 wives, possibly in 4 different places, and it is not unusual that one wife takes care of children of another wife, or of a sister, brother, in-laws, cousins or neighbors. For economic reasons people can be "registered" in 2 or even more places also in connection with landreform regulations and distribution of foodstuffs. All of this makes them reluctant to be explicit about their real dwelling place, and often they have no permanent real place of residence. Because of this very complex situation it is impossible to divide people in urban and rural, but this does not matter very much, because many of them undergo the influences of urban as well as of rural life, which possibly cancel one another out. Small children are of course worst off in this type of arrangement, which is further explained in the chapter on Xerophthalmia.

Insistent questioning is often resented by the people as they fear that the information will be passed on to various kinds of authorities and because they do not understand why a doctor should ask questions, instead of curing the patients at first sight, if possible even without examining them. History taking is therefore an arduous undertaking and probably some of the data collected in this way are not correct : from sheer politeness people often try to guess what the doctor likes to hear and will adapt their answers accordingly.

It is essential of course that the person who extracts the information knows the local language, and is patient enough to keep on with the conversation or desists for the time, if that seems better. Generally speaking, parents appreciate interest in their children, and will like to tell everything about them. But data concerning their own age, work, place of dwelling etc. they often try to avoid giving.

A number of blind patients is accompanied by non-relatives, neighbors etc. For that reason it is often not possible to get a history of any value : the blind person is not able to give information and the attendant often has not known him at the time of the blinding disease. It seems that a number of blind people is taken care of on a temporary basis only : staying a few months with one relative, them being passed on to another as the economic burden becomes too heavy. The amount of misery that can be guessed at, from these sparse data is staggering.

The "facts" which are stated can be surprising: a patient with enormous leucomata on both eyes says that it was caused by itching. No other signs or symptoms are remembered. A man from a distant village with large corneal scars is convinced that this was caused by an evil spirit, also no other information could be elicited.

Age of blind people.

The only kind of registration of the population which is compulsory is that people areto carry identification cards. The cards are issued at the government office nearest their dwelling place, but there is no hindrance to having 2 different cards issued, one in town and one in a village. This system was not yet perfected at the time of investigation 1958 - 1962. We had to rely for ascertaining the age of the patients on what they told us, most of the time, and could only correct it if they had stated an age which quite obviously did not agree with appearences. In the report on the data on age later on (chapter six) some of the consequences of this uncertainty will be explained.

Establishing of causes of blindness.

Generally the lack of correct data makes the group of "undetermined cause" much greater than could be wished. Fortunately from the type and site of the affection in a number of cases the cause can be guessed (e.g. fistula corneae in this area is very often caused by conjunctivitis gonorrhoica). Therefore an investigation of this type can only be carried out by an ophthalmologist with sufficient experience in the area itself, who can make an "educated guess" as to the real cause of blindness in a given case.

Mortality of young blind children.

If one looks at the curve of the age of blind persons as they come to the Outpatients' Dapartement, there seems to be a "lost generation". There are many small children who are blinded before the age of 3 years, there are far less children between the ages of 5 and 15, and afterwards the curve slowly rises again. It was because of this phenomenon that we instituted a follow-up examination into the fate of young blind children, which is reported in a special chapter, and which was carried out with financial aid from the National Research Council. It became claar that a rather large percentage of young blind children had died inside a few years after going blind, probably in part in connection with the disease which blinded them (malnutrition, neurological disease). At the time of the 1958 — 1962 investigation there was as yet no compulsory education : parents with a blind child would not bring it again for examination if they knew from a former visit that the child could not be helped. The number of blinding diseases which strikes at school age, and which would provide fresh cases, is very small. The question of how many blind children there really are is quite important as soon as compulsory schooling will be enforced because it will have to be known how many schools there will be needed with special facilities for blind and weak-sighted children. Until now they are just kept home by their parents, with the few exceptions who go to a school for the blind..

Time of seeking treatment.

If the patient is a child it is mostly recently blinded, but in the case of old people the reverse is more usual. Older people wait often for years in spite of very low vision before seeking help from a doctor. Living at a great distance, the cost of travel can be the reason, but more often they are "afraid" of modern medicine, or they have been told by people who are looked on as experts, that the doctor will do nothing before the patient is completely blind. Cataract is a wellknown disease, but the village expert does not know anything about either glaucoma or optic atrophy, which are quite common also. By this wrong kind of advice in many patients the right time for help passes unused, and if they are brought in at last, nothing can be done any more. If they have cataracts, it is often astonishing to see how even old people quite calmly accept the idea of an operation and submit to it without hesitation. It looks as if the problem had been talked over at home quite thoroughly, and if the long journey is made, there is no more doubt in their minds,. On the other hand there are some people who steadfastly refuse any kind of operation, and our patients who are classified as "blind by cataract" belong in this group. It can happen also that surgery is refused at first, but the patient returns after a few weeks and gives his permission : chances are that tbey have consulted a dukun (herb doctor) in between, which the doctor often hears afterwards from the nurses. Fortunately some of these dukuns give the same advice as the doctor!

Prevention.

From everything related above it is clear that a certain amount of guesswork is unavoidable. Common sense and experience must take the place of hard facts. In the evaluation of this type of investigation this should be taken into account, but still, in spite of reservations, it remains the only basis for further investigative work, and more important, for preventive measures. The latter are highly necessary and urgent, as it will be shown that a large part of the blinding diseases are preventable, part of them even at a not prohibitive cost. Especially as it seems probable that more of the blind young children survive than in former times, the case of the prevention of blinding diseases at an early age should not be put off until later, as that would increase the number of blind persons considerably...... and unnecessarily.

Chapter five.

Frequency of blindness in and around Surabaja, East Java.

For the investigation under report all patients who came to the Eye Hospital Undaan, Surabaja, Outpatients' Department were taken into account as far as they finally had visual acuity of less than $1^1/_2/60$ in both eyes. Two groups of blind people were investigated.

1. All blind patients seen at Undaan between 1-1- '58 and 1-1-'63.
2. All patients, who became blind finally, seen between 1-8-'66 and 1-8-'67.

In the chapter on "Possibilities for Keratoplasty" a smaller third group is considered which was seen between 1-1-'65 and 1-7- '65.

In this way samples covering almost ten years were studied.

Examination of patients.

All patients were seen by my colleague dr. Moh. Basoeki or myself, qualified ophthalmologists. Notes were taken of :

Name, age, sex, address, kind of work if any, status of each eye, probable cause of blindness.

According to the manual for use of the International Standard Classification it is essential to ascertain the exact age of beginning of blindness in each eye, but this was in many of our patients an impossibility. It was only possible to get an answer in ca. 30% of cases, and even then it could be the wrong answer ; as this number was too small to base any conclusion on it, this aspect of the investigation was left out of account.

Coding performed according to the International Standard Classification.

At the end of each month this was done by me with the help of lists of code numbers, which Mrs. Schappert-Kimmijser sent me at the time, although the Classification as such was not yet accepted as official. By doing this work soon after the examination it was often possible to complete them by having recourse to a new examination of the patient. Inconsistencies in coding could easily be ironed out and the same process was repeated once again at the end of each year.

To prevent double entry of the same patient, alphabetical lists were made, and before a patient was entered in the register it was checked if his or her name was already on our lists. This was not a useless measure, as there is a habit among the blind population of changing their place of living frequently, and people will not say in the first place, that they have been examined before, possibly for fear that this will influence the result of the examination. Blind persons who have been sent on from one relative to another do not even tell them, that they have been examined already, and are brought again from the new address. Sometimes if I recognized a patient he had even changed his name, which is not an unusual procedure on entering a new phase of life (marriage, other job) and which also seems to be tried out as a curative measure (?) when a person goes blind. The alphabetical lists cannot prevent that some people who have thus changed their name are entered twice, but every possible care is taken to prevent it.

Only patients who were definitely blind according to the definition were entered, people who were only temporarily blind during treatment did not enter. Mrs. Go — Sennema (= Seniman) took also the patients who were for the time being blind only, into account, giving both groups of data. As a means of judging the effect of treatment it has its uses, but as a final result only the smaller group has to be considered as "blind" and only this group can be compared with figures in the present study.

Different limit of visual acuity.

To be able to judge what would be the effect of a higher limit of visual acuity for the definition of blindness, there were made parallel lists during the year between Aug. last '66 and Aug. lst '67, one list contains the usual group, i.e. people who have a visual acuity of less than $1^1/_2/60$ in the best eye ; the other group (which naturally includes this group also) is based on a visual acuity of less than 1/10, which brings it in accordance with "Western" usage. In the same period also the number of people was considered, who had one good eye, whereas the other was blind, either according to the lower limit of visual acuity, or to the higher limit. In that way it was possible to see :

1. How large would be the difference if instead of the lower limit of visual acouity a more modern, higher one would be used.
2. How many people are blind in one eye only, according to both definitions of blindness.

Purposely the one-eyed patients were not mixed with wholly blind ones as only the latter group is important from a socio-economic viewpoint.

In the period Aug. 1st '66 — Aug. 1st '67 there were seen :

120 children under 17 yrs. of age, blind, visual acuity less than $1^{1}/_{2}/60$.

150 children under 17 yrs. of age, blind, visual acuity less than 1/10.

In the same period there were seen :

226 men and 222 women, over 16 yrs of age, visual acuity less than $1^{1}/_{2}/60$.

403 men and 401 women, over 16 yrs. of age, visual acuity less than 1/10.

For children 25% of the number of blind according to the lower limit has to be added to get an estimate of the number of blind by the higher limit. For adults : the number of blind men by the lower standard has to be increased by 78% to estimate the number of blind men according to the higher standard. For women this percentage is even 84%.

Frequency of blindness in the population visiting the outpatients' department. During the years 1958 through 1962 there were seen at the outpatients' department a total of respectively :

1958	18.243	patients, blind	1.014	=	5.5%
1959	18.553		990	=	5.3%
1960	19.219		1.006	=	5.2%
1961	18.020		865	=	4.8%
1962	17.768		825	=	4.6%

Aug. 1st '66 — Aug. 1st '67 : 13.965 patients, blind 568 = 4.8%.

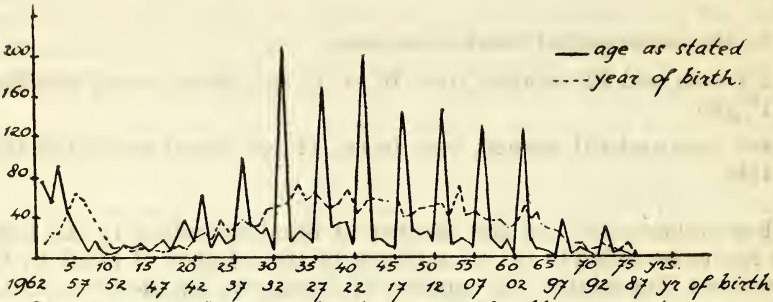
The incidence of blind patients among the population seen at the Outpatients' Department is rather stable, with a slight tendency only to decrease during the 5 first years which decrease did not keep up in the later period.

For a comparison with the population as a whole there is first needed a calculation of the incidence in various age groups.

Chapter Six.

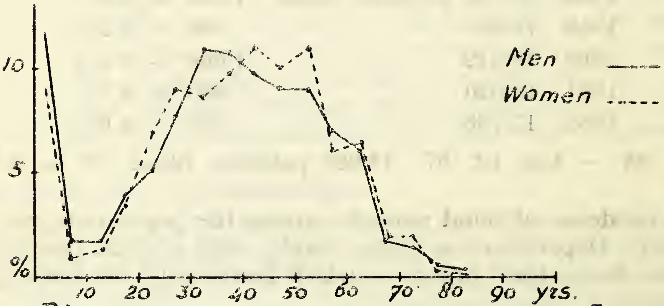
Age of blind patients at the time of examination.

From the data collected at the time of examination of the blind patients, seen at Undaan in 1958 through 1962, age was one which was always stated, but could not be verified. There is no official registration of the population, and people are not yet in the habit or memorizing their year of birth. Therefore the figures stated for age tend to be "round", i.e. the age is more often given as 35 or 40 than as 37 or 43, for example. In the Table 1. the age as stated is given, and if these figures are plotted in a curve, there appear sharp peaks every 5 years. As the whole investigation covered 5 years, these peaks cancelled one another out, if the year of birth was computed and plotted in a curve. (Fig. A) However a far better curve was obtained if the totals for 5-year periods were calculated (Table 3 and curve. B.) From this curve it could be seen that:



Blind men Undaän, 1958-1962. Comparison of Tables 1 & 2 Age in years and year of birth. Totals per year.

Fig. A.



Blind persons, Undaän 1958-1962, in 5-yr age groups, as a percentage of total of same sex. Table-3.—

Fig. B.

a) there is a very high figure for small children, even before the age of 3 years.

b) the figures for children between 6 and 16 years are much lower.

c) after the age of 16 the curve slowly rises, quicker and steeper for men, where it reaches a peak between 30 and 35 years of age, later and slower for women where the peak is reached between 40 and 45 years of age.

d) after 55 years of age the figures decrease again.

TABLE 1.

Age of blind patients, seen at Undaan 1958 through 1962, as stated by themselves at time of examination. Visual acuity less than 1.5/60.

Age	Male	Female	Age	Male	Female
— 1 yr	73	47	— 42 yr	16	11
— 2	54	59	— 43	16	16
— 3	91	52	— 44	9	12
— 4	42	38	— 45	8	5
— 5	31	19	— 46	144	199
— 6	16	4	— 47	24	19
— 7	9	4	— 48	21	11
— 8	12	7	— 49	22	16
— 9	5	9	— 50	15	10
— 10	4	5	— 51	167	232
— 11	9	9	— 52	14	13
— 12	8	5	— 53	17	10
— 13	12	7	— 54	14	11
— 14	5	4	— 55	8	7
— 15	13	7	— 56	129	119
— 16	16	16	— 57	15	11
— 17	8	10	— 58	9	3
— 18	20	17	— 59	16	10
— 19	35	29	— 60	7	6
— 20	19	11	— 61	129	133
— 21	62	75	— 62	17	10
— 22	13	18	— 63	8	10
— 23	25	38	— 64	4	4
— 24	13	18	— 65	1	4
— 25	17	16	— 66	36	33
— 26	99	132	— 67	3	5
— 27	34	19	— 68	6	2
— 28	22	24	— 69	3	3
— 29	30	24	— 70	—	2
— 30	12	13	— 71	30	38
— 31	208	162	— 72	3	2
— 32	21	10	— 73	2	3
— 33	26	14	— 74	1	3
— 34	15	6	— 75	3	—
— 35	10	4	— 76	5	4
— 36	166	166	— 77	2	—
— 37	27	12	— 78	2	1
— 38	29	10	— 79	1	1
— 39	34	25	— 80	—	—
— 40	17	6	— 81	6	3
— 41	196	231			

TABLE 2.

Year of birth of blind patients, seen at Undaan 1958 through 1962, computed from age as stated at time of examination. Limit of visual $1^{1}/_{2}/60$.

Year	Male	Female	Year	Male	Female
1881	4	2	1922	53	58
1882	4	3	1923	40	38
1883	2	—	1924	68	39
1884	1	1	1925	54	45
1885	2	3	1926	45	39
1886	2	—	1927	50	46
1887	4	1	1928	61	39
1888	2	4	1929	55	31
1889	13	10	1930	74	51
1890	4	12	1931	52	51
1891	9	9	1932	50	47
1892	12	14	1933	48	54
1893	5	10	1934	46	39
1894	6	5	1935	34	47
1895	13	13	1936	29	38
1896	14	10	1937	36	41
1897	14	11	1938	31	34
1898	25	27	1939	24	41
1899	27	33	1940	33	36
1900	41	49	1941	18	23
1901	34	23	1942	24	18
1902	47	37	1943	21	14
1903	33	25	1944	9	19
1904	29	27	1945	12	9
1905	36	29	1946	9	13
1906	37	30	1947	8	3
1907	38	35	1948	8	10
1908	43	41	1949	8	3
1909	41	74	1950	10	7
1910	72	66	1951	7	10
1911	31	47	1952	11	4
1912	51	57	1953	9	4
1913	47	37	1954	14	14
1914	45	51	1955	27	24
1915	43	62	1956	43	26
1916	36	60	1957	58	34
1917	36	44	1958	61	32
1918	56	47	1959	47	28
1919	54	59	1960	26	32
1920	57	71	1961	25	23
1921	56	51	1962	12	15

TABLE 3.

Age of blind patients, seen at Undaan 1958 through 1962, in 5 yr. intervals. as stated at first examination, and calculated as percentage of total number of patients of same sex. Visual acuity less than 1,5/60.

Age group	male	%	female	%
0 — 4 yrs	291	11,7%	215	9%
5 — 9	46	1.8%	24	1%
10 — 14	47	1.8%	32	1.3%
15 — 19	98	3.9%	83	3.5%
20 — 24	130	5.2%	165	7%
25 — 29	197	7.9%	212	9%
30 — 34	280	11 %	196	8.2%
35 — 39	273	10.9%	229	9.6%
40 — 44	245	9.8%	275	11.1%
45 — 49	226	9 %	255	10 %
50 — 54	220	9 %	273	11.1%
55 — 59	176	7 %	149	6%
60 — 64	159	6 %	161	6.5%
65 — 69	48	1.8%	45	1.9%
70 — 74	39	1.3%	46	1.9%
75 — 79	10	0.4%	6	0.25%
80 — 84	6	0.2%	3	0.12%

Blind Patients seen between 1/VIII — '66 and 1/VIII —'67 at Undaan.

From the total number of 520 men with visual acuity less than 1/10. 304 saw less than $1^1/_2/60$.

From the total number of 465 women with visual acuity less than 1/10, 286 had visual acuity less than $1^1/_2/60$. Only the last group was comparable with the group of patients seen in 1958 through 1962, but from the next Tables it is to be seen, that as far as regards age at the time of examination, there is no difference between the group with visual acuity less than 1/10 and that with less than $1^1/_2/60$.

TABLE 4.

Age of patients seen at Undaan 1/VIII-'66 — 1/VIII-'67 as stated at time of examination. Visual acuity less than $1^1/_2/60$ and less than 1/10.

Age	1/60	5/60	1/60	5/60	Age	1/60	5/60	1/60	5/60
	Male		Female			Male		Female	
— 1 yr	7	8	10	11	— 46	20	26	22	31
— 2	18	19	8	8	— 47	2	3	1	3
— 3	10	12	11	12	— 48	2	4	3	4
— 4	11	12	6	7	— 49	—	1	3	3
— 5	2	3	5	5	— 50	—	1	3	3
— 6	4	4	1	1	— 51	19	32	29	58
— 7	1	2	3	3	— 52	2	2	1	2
— 8	1	2	2	2	— 53	3	8	2	4
— 9	—	—	1	2	— 54	2	3	—	2
—10	1	1	—	—	— 55	1	1	1	1
—11	1	2	1	4	— 56	20	36	17	25
—12	1	2	2	3	— 57	3	6	—	1
—13	—	1	4	6	— 58	1	2	3	5
—14	1	1	2	3	— 59	1	1	2	2
—15	—	—	1	2	— 60	1	1	—	5
—16	1	4	2	5	— 61	12	30	10	26
—17	—	1	2	3	— 62	2	3	2	5
—18	2	3	1	3	— 63	2	4	3	4
—19	2	3	4	5	— 64	2	2	—	—
—20	1	3	—	2	— 65	—	2	1	4
—21	4	6	5	9	— 66	4	14	8	15
—22	1	2	—	1	— 67	4	6	3	3
—23	1	4	—	5	— 68	2	3	2	4
—24	4	5	1	3	— 69	3	5	1	1
—25	1	2	1	2	— 70	—	—	—	—
—26	3	6	11	18	— 71	5	14	1	5
—27	2	5	3	8	— 72	—	1	—	—
—28	3	7	2	5	— 73	—	2	1	2
—29	3	5	2	2	— 74	1	1	—	—
—30	1	2	1	1	— 75	1	2	—	1
—31	21	34	23	36	— 76	5	8	2	6
—32	1	3	1	2	— 77	—	1	—	—
—33	3	6	3	3	— 78	—	2	—	1
—34	3	5	1	2	— 79	1	2	—	—
—35	1	2	—	2	— 80	—	—	—	—
—36	20	28	19	37	— 81	3	9	2	5
—37	5	6	1	2	— 82	—	—	—	1
—38	2	4	2	2	— 83	—	—	—	—
—39	4	6	2	2	— 84	—	—	—	—
—40	2	2	2	2	— 85	1	1	—	—
—41	16	41	26	41	— 86	—	—	1	1
—42	3	4	—	—	— 87	—	—	—	—
—43	3	3	—	—	— 88	—	—	—	—
—44	3	3	—	1	— 94	—	—	—	1
—45	4	7	1	3	—101	—	1	—	—
					Total	304	520	286	465

As the above Table only covers one year, the peaks at the round figures are very noticeable again. If we calculate the figures for 5-year age groups, as in the case of the 1958/1962 figures, we can see that the curve is in principle the same. It shows a high figure for blind young children, even higher than in the years 1958 through 1962. After 16 years of age the rise in number of blind patients starts somewhat later, as well for women as for men. This means that the situation har improved because there are less blind people of working age than before, as compared to the total number of blind patients.

TABLE 5,

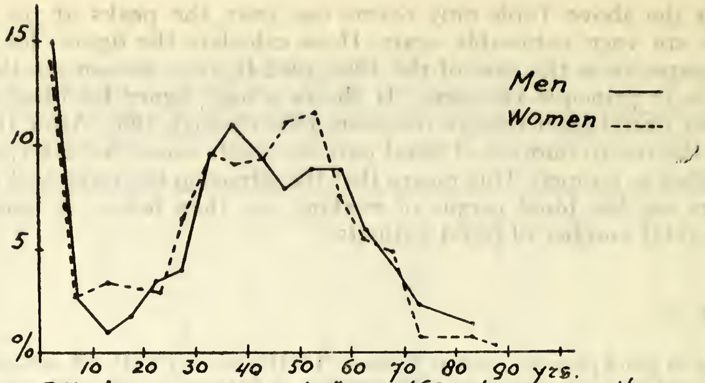
Age of blind patients, seen at Undaan, 1/VIII-'66 — 1/VIII-'67, in 5-year age groups, and as a percentage of total number of patients of the same sex. Limit visual 1/60 and 5/60.

Age	1/60	% Male	5/60	%	1/60	% Female	5/60	%
0 — 4 yr	48	15 %	54	10 %	40	14 %	43	9 %
5 — 9	7	2.3 %	9	1.7 %	7	2.8 %	8	1.7 %
10 — 14	3	1 %	6	1.1 %	10	3.5 %	18	3.6 %
15 — 19	6	1.9 %	14	2.7 %	9	3.1 %	18	3.6 %
20 — 24	11	3.6 %	19	3.6 %	7	2.8 %	20	4.2 %
25 — 29	12	3.9 %	25	4.8 %	19	6.6 %	34	7.2 %
30 — 34	29	9.5 %	50	9.6 %	28	9.7 %	45	9.6 %
35 — 39	33	10.8 %	46	8.8 %	26	9 %	45	9.6 %
40 — 44	29	9.5 %	58	11 %	27	9.4 %	44	9.4 %
45 — 49	24	7.8 %	35	6.5 %	32	11 %	67	14 %
50 — 54	27	8.8 %	46	8.8 %	33	11.5 %	38	8.1 %
55 — 59	27	8.8 %	46	8.8 %	22	7.6 %	39	8.3 %
60 — 64	18	5.9 %	41	7.8 %	16	5.6 %	23	5 %
65 — 69	13	4.2 %	28	5.3 %	14	4.9 %	8	1.7 %
70 — 74	7	2.3 %	20	3.8 %	2	0.7 %	7	1.5 %
75 — 79	6	1.9 %	13	2.5 %	2	0.7 %	6	1.2 %
80 — 84	4	1.3 %	10	1.8 %	2	0.7 %	—	—
85 — 89	—	—	—	—	1	0.3 %	1	0.2 %
90 — 94	—	—	—	—	—	—	1	0.2 %
95 — 99	—	—	—	—	—	—	—	—
100—104	—	—	1	0.2 %	—	—	—	—
Total	304	100 %	520	100 %	286	100 %	465	100 %

From the Table 5 a curve can be plotted, to compare the influence of the visual limit of 1/60 with that of 1/10 on the incidence of blindness with age : there are in principle no differences. The curves are almost identical. (Fig. C and D)

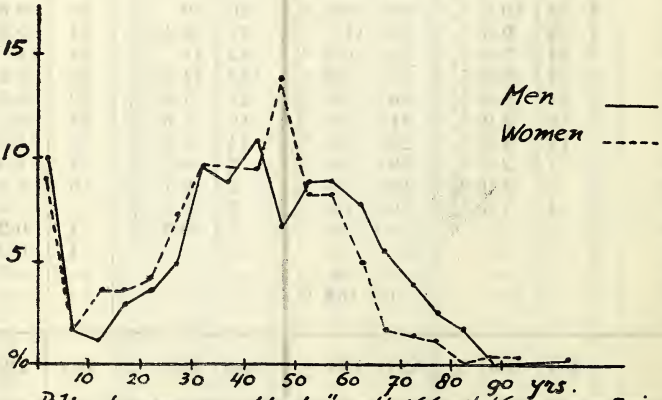
Comparison with the division of age groups of an average population.

From the data of an investigation done in Tjermee/Duduk Sampejan, a rural area not far from Surabaja, which can be considered as typical, a curve was plotted in the same way as the curves for percentage of blindness with age. The data were collected and published by Halvor



Blind persons, Undaän 1/8'66 - 1/8'67, acuity less than 1½/60, in 5-yr age-groups, as a percentage of total of same sex, Table-5. —

Fig. C



Blind persons, Undaän 1/8'66 - 1/8'67, in 5-jr. age groups as a percentage of total of same sex. Acuity less than 1/10. Table-5. —

Fig. D

Gille & dr. R.H. Pardoko (148). They are normal as compared to similar figures from the 1961 census for rural areas in Java.

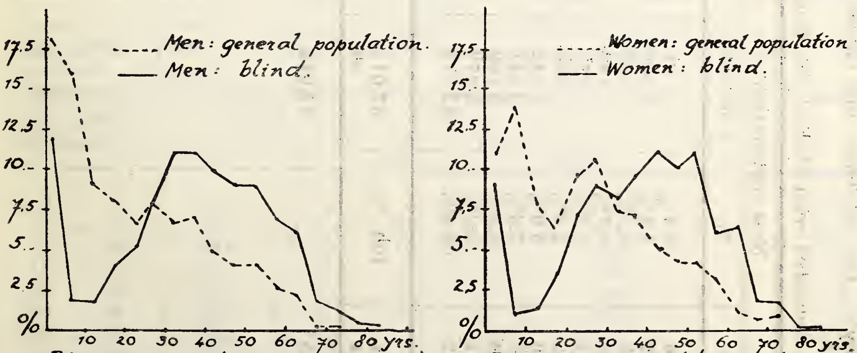
Population curve, in 5 year age groups, calculated as a percentage of the whole sample of the same sex taken as 100. 1961.

TABLE 6.

Age group	Jen	Women
0 — 4 yr	18 %	11 %
5 — 9	16 %	14 %
10 — 14	9 %	7.7 %
15 — 19	8 %	6.4 %
20 — 24	6.5 %	9.7 %
25 — 29	8 %	10.6 %
30 — 34	6.5 %	7.2 %
35 — 39	7 %	7.1 %
40 — 44	4.7 %	5.1 %
45 — 49	4 %	4.2 %
50 — 54	4 %	4 %
55 — 59	2.8 %	3.2 %
60 — 64	2.4 %	1.1 %
65 — 69	1.1 %	0.9 %
70 — 74	1.1 %	1.1 %

The losses in the age groups between 10 and 20 years of age concern children who have been born in the years of occupation and revolution, at which time probably the care for small children was very difficult causing a high mortality in these groups.

Otherwise, if the curves for percentages of blindness with age (B&C) are compared with the normal distribution of the population over the age groups, one can see, that the incidence of blindness is high at a very early age, and then again after the age of 30 it rises above the expected incidence and stays higher until over the age of 70 years. (Fig. E)



Blind men in %/decade (Table-3). compared to men in general population in %/decade (Table-6).

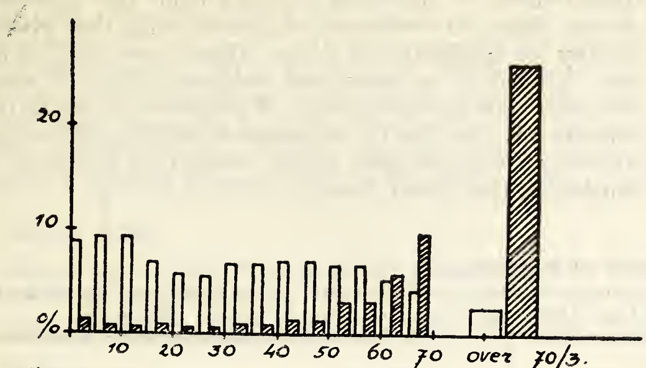
Blind women in %/decade (Table-3) compared to women in general population in %/decade (Table-6).

Fig. E

TABLE 3 from Sorsby's „The Incidence and Causes of Blindness in England and Wales 1948 - 1962" 1966 p.5. New registrations 1955 - 1962.

Age distribution compared with that of the enumerated population at the 1961 census.

Age group	New registrations				Persons		Census 1961		Persons
	Male	%	Female	%			Male	Female	
0 — 1 yr	146	0.4%	81	0.1%	227	0.3%	8.3%	7.3%	7.8%
1 — 4	482	1.4%	369	0.7%	851	0.9%			
5 — 15	503	1.5%	332	0.6%	835	0.9%	17.5%	15.6%	16.6%
16 — 20	243	0.7%	140	0.2%	383	0.4%	7.0%	6.5%	6.7%
21 — 29	437	1.3%	271	0.5%	708	0.8%	11.7%	10.8%	11.2%
30 — 39	885	2.6%	536	0.9%	1.421	1.6%	14.0%	13.1%	13.5%
40 — 49	1.356	3.9%	1.142	2.0%	2.498	2.7%	13.8%	13.4%	13.6%
50 — 59	2.592	7.6%	3.141	5.5%	5.733	6.3%	13.4%	13.4%	13.4%
60 — 64	2.123	6.2%	3.109	5.4%	5.232	5.7%	4.9%	5.7%	5.3%
65 — 69	3.302	9.6%	5.273	9.3%	8.575	9.7%	3.7%	4.9%	4.3%
70 & Over	22.232	64.8%	42.619	74.8%	64.851	71.0%	5.7%	9.4%	7.6%
	34.301	100 %	57.013	100 %	91.314	100 %	100 %	100 %	100 %
Unknown	34		59		93				
	34.335		57.072		91.407				



Sorsby's Table 3. White: general population in %/decade, census 1961, black: blind persons in %/decade, new registrations 1955-1962.

Fig. F

Table 7. Peroentages of blind persons registered by age groups and sex. Undaan, Surabaja, 1958 through 1962. Visual acuity less than $1^1/_2/60$. (Compare Table 5, Sorsby, 1966, p. 7.)

	0—17 yrs	%	16—41 yrs	%	cver 40 yrs	%
Male	402	16 %	1.139	45 %	942	38 %
Female	298	13 %	1.091	45 %	991	41 %
Total	700	14 %	2.230	45 %	1.933	39 %

Table 5 from Sorsby "The incidence and Causes of blindness in England and Wales 1948-1962". 1966, p. 7. Age at registration shown in certificates of blindness, 1955 — 1960. All ages.

Age groups in years	Males		Females		no.	Persons	%
	no	%	no	%			
0 — 1	100	0.4 %	59	0.1 %	159		0.3%
1 — 4	346	1.5 %	255	0.7 %	601		1.0%
5 — 14	294	1.3 %	192	0.5 %	486		0.8%
15 — 19	175	0.8 %	106	0.3 %	281		0.4%
20 — 29	315	1.4 %	193	0.5 %	508		0.8%
30 — 39	638	2.7 %	381	1.0 %	1.019		1.7%
40 — 49	941	4.0%	733	1.9%	1.674		2.7%
50 — 59	1.720	7.4%	2.086	5.4%	3.806		6.2%
60 — 69	3.499	15.0%	5.559	14.5%	9.058		14.7%
70 — 79	7.543	32.4%	13.161	34.3%	20.704		33.6%
80 — 89	7.015	30.2%	13.443	35.1%	20.458		33.2%
90 — over	679	2.9%	2.175	5.7%	2,854		4.6%
	23.265	100 %	38.343	100 %	61.608		100 %
not stated	205		315		520		
	23.470		38.658		62.128		

COMMENT : The division of the blind patients over the various age groups shows an enormous difference with that given by Sorsby for England and Wales. There over 65% of blind men is aged 70 or over and even over 75% of women, whereas here the percentage of people who are so old is already low, but also the incidence of blindness has its peak at a much younger age : which means that it is more of a burden on the labour force.

Chapter seven.

TABLE 8. Degree of blindness.

Percentage blindness (visual acuity =O in both eyes) by age and sex. Undaan, 1960 only, (Compare Table 6. Sorsby, 1966, p. 9).

	0-17 yrs	16-41 yrs	over 40 yrs
Male	48 %	16.3 %	13.7 %
Female	40 %	25.5 %	25.2 %

TABLE 9. Percentage of persons by specified causes who were totally blind (visual acuity = 0 on both eyes). Undaan. 1960 only, by age groups.

Cause	0 — 17 yrs	over 16 yrs.
12. Gonorrhoea	57 %	23.2%
18. Variola	—	50 %
19. Syphilis	25 %	15.5%
20. Trachoma	50 %	7.5%
22. Typhoid	100 %	—
28. Other infection	—	33 %
65. Neurological	80 %	100 %
67. Malnutrition	45.2%	100 %
89. Congenital	16 %	—
91. Unknown to science	100 %	28.6%
98. Unknown, Wr —	60 %	16 %
99. Unknown	60 %	35 %

From Sorsby "The Incidence and Causes of blindness in England and Wales 1948 - 1962". 1966,, Table 6.p.9. Degree of blindness by age groups and sex : percentage distribution. (in part).

All ages

	M	F	P
number on which information is available	23.173	38.242	61.415
Percentages.			
No light perception	3.6%	3.2%	3.4%
Light perception	11 %	10.1%	10.4%
1/300 — 3/60	56.9%	60.0%	58.8%
Over 3/60	28.5%	26.7%	26.7%
	100 %	100 %	100 %

Comment : Only for the year 1960 there was made a specification of the percentages of patients who had no visual perception at all (visual acuity = 0 in both eyes) compared with blind people who had some visual rest, but not more than $1^1/_2/60$ in the best eye. Table 8 gives the percentages. Relatively more children are totaly blind, which is the same in England, but for adults it can be said that the blind in East Java are "blinder" than those in England in our investigation. This is probably related to the type of blinding diseases which is also quite different in both regions.

Chapter eight.

Manual on the Use of the Standard Classification of Causes of Blindness for international Use.
(Journal of Social Ophthalmology, hors série, 1966, Paris.).

Preface.

The purpose of this internasional standard classification is to achieve a uniform classification of the causes of blindness so that comparable material can be obtained all over the world.

In behalf of a good and uniform use the scheme is preceded by some explanations and instructions and followed by an index of diagnostic terms indicating etiology, and by another indicating site and type of affection.

Finally a list of some clinical entities, causing blindness, supplied with the two code numbers has been added.

Explanation of the classification.

The outstanding characteristic of this standard classification is that it requires twofold classification of causes of blindness. Each case must be classified with respect to both the etiological factor and the site (topography) and type (nature) of the eye affection which has resulted in blindness.

In both classifications the different causes have been placed in significant main divisions. A main objective has been to keep the classification sufficiently condensed to facilitate its use without loss of important detail. Provision has been made for future subdivisions within these larger groupings where they are found desirable. -p-6-

Code numbers.

For convenience in tabulation a code number has been assigned to each diagnosis in both classifications. For the etiological factor, numbers under 100 are used, decimals being employed only when detailed subdivision is necessary (or desirable). For the items by site and type of eye affection, numbers over 100 are assigned.

Medical terminology.

The medical terminology used in the standard classification will be found to conform to that commonly used by practising ophthalmologists in recording eye examinations of blind persons.

Experience with the classification seems to indicate that the simple terminology adopted tends to promote accuracy in the preparation of statistics. Moreover it has the advantage of being readily translated in programs of public education.

Use of classification.

Definition of blindness.

Since the aim is to produce comparable statistics, it is recommended that the tabulation should be confined to cases coming within the same definition of (economic) blindness.

Economic blindness is loss of sight to such a degree that the individual is unable to engage in occupations for which eyesight is essential.

However the criteria for this sort of blindness often vary in the different countries. It should therefore be made entirely clear in the report of the study, which limits for economic blindness are accepted in order that due allowance may be made in attempting comparisons or combinations of data.

Persons who are blind only in one eye are not included in the above definition.

Blindness of the two eyes from different causes.

In most cases of blindness, the causes will be found to be the same for each eye. In the relatively small number of cases in which the causes for the two eyes are substantially different, it is recommended that only the cause affecting the second eye to become blind be included in the main tabulation. This procedure throws emphasis on the cause of the economic blindness of the individual.

It may however be desired to accumulate data on persons with different causes of blindness in the two eyes, in which case the number of such cases should be recorded, and supplementary tabulation made of the cause of blindness in the first and second eye affected.

Classification by multiple causes.

Each case should only be classified once under each heading. In those cases in which it is recognized that more than one factor has contributed to blindness it is desirable that the most important causes should be tabulated,

Classification by primary cause.

All cases should be classified by the primary cause, not by a secondary affection, which may be the ultimate cause of the blindness. The physician making the diagnosis, should therefore be requested to indicate clearly his judgement as to the primary eye lesion and its primary etiology. If information regarding secondary causes is required, such data should be presented separately as a supplementary table.

Physician's report on eye examination.

Statistical use of eye examination records will be facilitated if the ophthalmologist's diagnosis is originally recorded in the detail required by the classification.

Any form should include all of the following items, which are considered essential : separate recording of the age of onset of blindness for each eye; the etiology ; the eye condition which caused blindness ; secondary affections of the eye, if present ; the measurement for each eye of central visual acuity ; the measurement of the field of vision ; hereditary factors if present ; consanguinity of parents if present. It is highly desirable that record forms include also items on prognosis and the ophtalmologist's recommandations for reexamination and corrective treatment as well as basic social data such as date of birth, sex and race. Moreover it will be of value that the questionnaire also contains some questions on social circumstances of the patient and the possibility of rehabilitation.

It will be helpful if diagnoses are recorded in the terminology of the classification. It is recommended therefore, that public or private agencies intending to use eye records from a particular -p.8- group of ophthalmologists provide such ophthalmologists with copies of the classification and invite their cooperation in making records as complete and as convenient for tabulation as possible.

Although provision is made in each main division of the classification for including indefinite and unspecified items under "not specified" headings, the data as a whole will be of little value unless the proportion of these unsatisfactory items is relatively small. The examining ophthalmologist should be requested to record site and type of eye affection as definitely as possible, and to specify etiology whenever it can be determined. Some cases of undetermined causes must be expected, however, particularly when physicians are examining persons whose blindness has been of long duration. The desire to avoid "not specified" items should not lead to the encouragement of recording of hypothetical diagnoses.

Frequency of Cause of Blindness Tabulations.

It is suggested that studies of causes of blindness be made annually, or less often if the changes due to turnover of cases or to changes in the diagnostic information available appear too slight to warrant new tabulations. Preferably, the tabulations should include the entire group of blind persons under care or on the register of the agency making the study, in order that they may represent as nearly as possible the situation at the date of the report.

Tabulation of new cases of blindness occurring in a year will be of special interest only when, and if reporting of cases as they occur has been established. The count of new cases applying for service in any year would have no particular significance. They would not indicate the incidence of blindness from each cause during the interval covered by the report, since in any given case a period of years may elapse between the time of onset of blindness and the date of application for service.

Cross-tabulation of causes of blindness.

For scientific uses the most satisfactory method of presenting data on causes is in a cross-tabulation correlating the etiological factors with the site and type of eye affection.

Editing the eye examination records.

As is the case in compiling any statistics derived from case records supplied by many different -p.9- examiners, it is important that the eye examination records be carefully edited before any attempt is made to tabulate. This is essential, in order to insure that data reported are sufficiently complete and in proper form for tabulation, and that cases of a similar type will be found under the same classification in the figures from each group studied.

Types of records which will require action on the part of the editor are : Records which are incomplete.
Records on which the physician has ignored the individual items on the form and stated the facts in running comment.
Records on which one or more of the diagnostic items are obviously out of place. Records involving two or more commonly associated eye conditions which are given different emphasis by different examiners but which should be coded consistently.

Records on which the examiner has given a description of the eye conditions but no summary diagnosis.
Records on which the diagnosis, the description of the appearance of the eyes, and the degree of vision are obviously not in agreement.
Records on which the diagnosis only is given in etiological terms and site and type of eye affection have not been recorded.
Records on which the examiner lists two or more eye conditions as primary. It is extremely important that the preparatory work on the individual case records should not be delayed too long after the eye examination. If an effort is made to review each record for completeness and to edit and code it for statistical purposes at the time of its receipt from the examining physician, it will usually be possible to secure additional information needed, either by corresponding with the examining physician or by requesting an investigation of the history of the case.

Responsibility for editing.

If the research worker who is conducting the study is not himself an ophthalmologist, it is necessary that the services of a qualified ophthalmologist be provided for consultation concerning the study, particularly concerning the adequacy of the medical data used. Editing of the records should be done either by the person responsible for the study, or also by a specially trained assistant under his immediate supervision. Whoever takes part in the editing process must become thoroughly familiar with ophthalmological terminology and with the fundamentals of the classification before editing is undertaken. -p.10-

In the process of editing, all those records presenting any question concerning adequacy or correctness of the recorded diagnosis should be sorted out for examination by the advising ophthalmologist. He may complete or rearrange the information given on the record. Often this is a matter of interpreting the facts presented and can be done without consulting the examining physician. In certain cases, it may be desirable to delay final classification until further information on etiology is obtained from the examining physician, or from other reliable sources, such as the patients' previous physician.

Coding the records.

The task of assigning code numbers should be done by the person who edits the records as part of the editing process. To facilitate this task an index of diagnostic terms with the two code numbers has been prepared and is given on pp...............

RULES FOR APPLYING THE CLASSIFICATION BY ETIOLOGY

The etiological classification is determined by the disease or accident which is responsible for physiological or pathological changes in the eye, that is by the cause underlying the eye condition. The ideal ophthalmological examination includes such supplementary tests, examinations, and study of the case history as are necessary to determine the etiology.

Etiology should not be coded automatically.

Cases should not be classified automatically as resulting from the etiological condition which is assumed to be the most common cause of the given condition. For example, interstitial keratitis is not always caused by prenatal syphilis ; nor is all ophthalmia neonatorum gonorrhoeal. Each case should be classified on the basis of specific information which has been determined to apply in the case of the given individual.

Etiology unknown.

Theoretically, the etiological factor should be determinable in every case of blindness. Actually, it may be difficult and often impossible to establish the etiology in a particular case because of the gaps in present medical knowledge or the lack of adequate information concerning the history of the case.

Many cases in which the etiology is not given or -p-11- is noted merely as unknown should properly be classified (at least for the present) as "unknown to science", 91,0. For example :

Myopia cases, unless specified as of "hereditary origin" ;
Cataract (senile type), when no etiology is noted by the examiner and unless specified as of hereditary origin ;
Glaucoma, except congenital cases or those otherwise specified by the examiner as of hereditary origin ;
Pemphigus, unless or until more definitelely specified ;
Other case records on which the etiological item is omitted or designated as "unknown" should be classified as :
"Evidence insufficient for diagnosis", 98.0. if there is some indication that the examiner has made an unsuccesful attempt to determine the etiology ; or "No report on etiology" 99,0. if the record of the examination is obviously incomplete.
If the etiology can be only partially determined sometimes an indefinite category under the general grouping can be used e.g.
"infectious diseases not specified" 29,0 ;
"External causes of accidents, poisonings, violence not specified" 49,0 ;
"disorders of central nervous system not specified", 65,9 ;
"general diseases not elsewhere classified, not specified" 79,0 ;
"prenatal influence not specified", 89,0.

A diagnosis of "probably (or possibly) due to "some etiological factor may be definitely classified only if it is the examiner's best judgement on the case and there is no evidence that etiology has been assigned automatically.

Unsatisfactory diagnoses.

Some systemic diseases which result in blindness are unclassifiable unless the diagnosis is complete. For example :
Encephalitis and meningitis should be assigned to the primary etiological factor to which it is due (as an infectious disease 14.0, 19.2, 21, 18.0 ; traumatic 40,0 - 49.0). If this cannot be determined, it must be classified as a disease of the central nervous system 65,8.

Clues from other parts of the record.

Sometimes clues contained in other items on the record or on other records may suggest the -p-12-

etiological classification of a case. For example :
Cases in which the record indicates that blindness existed from birth should be classified as "prenatal influence not specified" 89,0 unless there is evidence on the record indicating that the blindness is of genetic origin 81,0 to 82,0 ; the result of a wellknown affection of the mother during pregnancy, 19,1. 25,1, 67,0 ; the result of injury during birth process, 45,0, or caused by an affection at birth 12,0, 28,0.
A case involving sympathetic ophthalmia in the second eye following a wound of the first eye, is attributed to the injury to the first eye and should be so classified.
If an eye already blind (as from structural anomalies) is later injured, the original cause should be coded.

Hereditary factors need special scrutiny.

If the physician's report shows an eye defect which is recognized as inheritable, the case should not be finally classified until information concerning the individual is supplemented by careful investigation of family history. Depending upon the conclusiveness of the evidence, such cases may be classified as :

"genetic origin established", 81,0, provided it is demonstrated that the same inheritable defect has appeared in other members of the family in accordance with the mode of transmission expected for the defect in question or that in the case of an inheritable defect consanguinity of parents exists.
"genetic origin presumed" 82,0, if the defect is one that is recognized as most often inheritable, but evidence from the family history cannot be produced.

This number includes the following affections : all primary tapetoretinal dysfunctions; macular dystrophy (Stargardt); macular aplasia; coloboma of the macula (if genuine) ; retinoschisis ; angioid streaks ; chorioideremia; total colour blindness; albinism; optic nerve atrophy Leber.

Some blindness occurring in more than one member of the same family may be due to the same cause but not to heredity. For example blindness at or soon after birth may be due to prenatal syphilis, 19,1 ; nutritional deficiency, 67,0 ; injury during birth process, 45,0 or to poisoning by dysoxygenation, 46,15.

Similarly, cases occuring in two or more related adults, may be due to infection or faults of nutrition.

Avoid classification of such cases under the genetic items.

RULES FOR APPLYING THE CLASSIFICATION BY SITE AND TYPE OF AFFECTION.

For this grouping each case should be classified according to the location in the eye (topography) -p-13-
and the nature of the lesion, responsible for the failure of sight. (e.g. inflammation, degeneration).

Multiple diagnoses.

In classifying cases having lesions in more than one part of the eye, careful consideration should be given to the following points :

If in the opinion of the examining ophthalmologist one lesion is secondary to the other, the case should be classified according to the primary lesion. (e.g. glaucoma secondary to cataract or vice versa ; glaucoma and/or cararact secondary to uveitis ; retinal detachment secondary to operation on a congenital cataract).

The classification includes items for the more frequent combinations (e.g. iridocyclitis, uveitis, kerato-iridocyclitis, sclero-iridocyclitis, chorioretinitis).

In cases having a number of concurrent lesions, anyone of which might result in blindness, try to select one of the general items in the classification, which will cover all lesions (e.g. classify the case of a person born with several structural anomalies under "multiple congenital anomalies" 147)

Microphthalmos is often associated with other structural anomalies of the eye. If the microphthalmos is very slight, the other anomaly is classified e.g. aniridia, 145 ; colobomata 146 ; congenital cataract 410. If the microphthalmos is obvious, the case is classified under "multiple congenital anomalies". 147. If the microphthalmos is the most obvious feature, the case is classified under microphthalmos 144.

Myopia is often found together with other causes of blindness (e.g. with tapetorinal degeneration, congenital cataract, colobomata, trachoma other affections of the cornea). Myopia is not taken as the cause of blindness in cases, if there is another important cause.

Other refractive errors should not be used in the coding, because they are not causes of blindness,

Uveitis, iridocyclitis or any other term implying inflammation of the second eye following injury to the first should be classified as "sympathetic ophthalmia", 570, unless the record indicates a different etiology for the second eye.

If in a case of injury more than one part of the eye is affected, the case is coded under "general affection of eyeball specified" 180.

Tapetoretinal dysfunction together with other affections of the eye or as part of a syndrome have to be classified under ,,tapetoretinal degeneration and allied conditions". 650.
(e.g. t.r.d. with colour blindness, with optic nerve atrophy and dislocation of the lens, with microphthalmos, with keratoconus, in congenital amaurosis Leber, in Oguchi's disease, in Biedl- Bardet's disease, in Spielmeyer-Vogt's disease).

Clues from other parts of record.

Clues to proper classification by site and type of affection are at sometimes found in other items beside -p.14-
the eye condition responsible for blindness. For example :

The fact that an individual lacks the ability to project light may lead to suspicion that cataracts are secondary to some other eye lesions. If there is no further information, the record should be returned to the examiner. The word "secondary" is sometimes used by an examiner in recording an eye condition of known etiology (e.g. "secondary" optic nerve atrophy due to brain tumor). The fact that no other eye condition is specified in the diagnosis as "primary" suggests that the lesion mentioned should be coded as the eye condition responsible for blindness.

Another confusing use of the word "secondary" occurs in the term "secondary cataract". This term is sometimes used to indicate that the remnants of a partially removed cataractous lens are present, in which instance the eye condition is to be coded as "cataract", 410.

Unsatisfactory diagnosis.

Occasionally the diagnosis will have been stated in terminology which is unacceptable, either because the term itself is indefinite or because blindness from the eye condition is unusual or improbable. These cases warrant further investigation before acceptance.

Terms which describe merely the sequellae of eye diseases should be avoided. For example:

Optic nerve atrophy (like heart failure) may be the final stage of many different conditions.

Cases in which the optic atrophy is not primary, but apparently followed another eye lesion of sufficient importance to cause blindness, should be classified by the other lesion.

The term "enucleation" by itself is inadequate as a diagnosis in that it gives no clue to the type of eye lesion that preceded it. If it is not possible to discover the actual cause of blindness, the case must be classified as "excised eyeball", 150.

Corneal opacities are not classifiable as such. If an eye disease such as corneal ulcers or interstitial keratitis has caused the opacity, this cause should be classified. If, however, it is not possible to tell from the etiology or the history of the eye condition whether the opacity is caused by such disease or by an injury, the case must be classified as "affection of the cornea, not specified", 390.

Certain terms suggest merely the symptoms or complications which occur in association with eye conditions causing blindness. For example :

Optic nystagmus is usually associated with imperfect vision. It is preferable, therefore, to classify by the eye condition primarily responsible for the low vision, if this is known (e.g. congenital cataract). However, if the case is one in which there is no visible eye defect or lesion, it should be classified as "congenital nystagmus", 950.

Strabismus, (squint, convergent or divergent) in early life may result in blindness or low vision, but this would affect one eye only. When it occurs in association with another eye condition that may cause blindness, the latter should be assumed to be primarily responsible for blindness of the individual.

If it is desirable to make a separate tabulation of the cause of blindness in the first and second eye affected, the blindness or low vision of the squinting eye is coded under "amblyopia ex anopsia", 940.

STANDARD CLASSIFICATION OF CAUSES OF BLINDNESS FOR INTERNATIONAL USE.

(International classification accepted by the General Assembly of the International Association for Prevention of Blindness at New-Delhi, on December, 4th, 1962).

I. BY ETIOLOGY.

Infectious diseases :

11.0 Diphtheria.
12.0 Gonorrhoea
13.0 Measles
14.0 Meningococcal infection
16.0 Scarlet fever
17.0 Septicemia
18.0 Smallpox
19.1 Syphilis prenatal
19.2 Syphilis postnatal
19.9 Syphilis, pre- or postnatal not specified.
20.0 Trachoma
21.0 Tuberculosis
22.0 Typhoid fever
23.0 Rubella
24.0 Onchocerciasis
25.1 Texoplasmosis, prenatal
25.2 Toxoplassmosis postnatal
25.9 Toxoplassmosis pre- or postnatal not specified.
26.0 Brucellosis
27.0 Leprosy
28.0 Other infectious disease specified
29.0 Infectious disease, not specified

Accidents, poisoning, violence :

I. Classification by external cause :

40.0 Occupational activity
41.0 Military activity
42.0 Household activity
43.0 Play or sport other than occupational
44.0 Traffic or transportation other than occupational.
45.0 Birth process
46.0 Surgical or medical procedure
47.0 Act of violence
48.0 Other external cause specified
49.0 External cause not specified

II. Classification by causal agent :

.01 Chemical causing burn
.02 Radiation
.03 Other agent causing burn
.04 Firearm using explosive

.05 Fireworks
.06 Other explosive
.07 Airgun or slingshot
.08 Other sharp or pointed object
.09 Blow or fall.
.10 Foreign body in eye
.11 Methylalcohol
.12 Dinitrophenol
.13 Lead
.14 Quinine
.15 Dysoxygenation
.18 Other causal agent specified
.19 Causal agent not specified

General diseases not elsewhere classified:

61.0 Anemia or other blood disease
62.0 Diabetes mellitus
63.0 Nephritis or other kidney disease
64.0 Vascular disease (including arteriosclerosis and other cerebro-vascular lesions)
65.1 Multiple sclerosis
65.2 Intracranial neoplasm
65.8 Other disorder of central nervous system specified
65.9 Disorder of central nervous system not specified
66.0 Complication of pregnancy
67.0 Nutritional deficiency
68.0 Sarcoidosis
78.0 Other general disease not elsewhere classified, specified
79.0 General disease not elsewhere classified, not specified

Prenatal influence not elsewhere classified:

81.0 Genetic origin established
82.0 Genetic origin probable
88.0 Other prenatal influence not elsewhere classified, specified
89.0 Prenatal influence, not specified

Etiology undetermined or not specified:

91.0 Unknown to science
98.0 Evidence insufficient for diagnosis
99.0 No report on etiology

II. BY SITE AND TYPE OF AFFECTION.

Eyeball in general:

110 Glaucoma excluding infantile
121 Myopia with detachment of retina
122 Myopia, detachment of retina not specified
130 Panophthalmitis and acute endophthalmitis
141 Albinism
142 Anophthalmos, excluding surgical
143 Hydrophthalmos (infantile glaucoma)
144 Microphthalmos

145	Aniridia
146	Coloboma, any part, excluding surgical
147	Multiple congenital anomalies
148	Other congenital anomaly specified
149	Congenital anomaly not specified
150	Disorganized eyeball (atrophic globe, phthisis bulbi, or excised eyeball)
180	Other general affection of eyeball specified
190	General affection of eyeball, not specified

Conjunctiva :

211	Purulent ophthalmia of newborn (ophthalmia neonatorum)
212	Purulent ophthalmia other than newborn
280	Other affection of conjunctiva specified
290	Affection of conjunctiva, not specified

Cornea :

311	Interstitial keratitis
312	Phlyctenular keratitis, including keratoconjunctivitis
313	Ulcerative keratitis, including trachoma
315	Leucoma adhaerens
316	Fistula corneae
318	Keratitis, other type specified
319	Keratitis, type not specified
320	Corneal dystrophy, degeneration
360	Keratomalacia
370	Keratoconus
380	Other affection of cornea specified
390	Affection of cornea, not specified

Lens :

410	Cataract
420	Dislocated lens
480	Other affection of lens specified
490	Affection of lens, not specified

Uveal tract :

510	Iritis, iridocyclitis or uveitis
530	Kerato-iridocyclitis or sclero-iridocyclitis
550	Choroiditis
560	Chorioretinitis
570	Sympathetic ophthalmia
580	Other affection of iris, ciliary body or choroid specified (including neoplasm)
590	Affection of iris, ciliary body or choroid, not specified

Retina :

610	Retinopathy, including retinitis and lesions of retinal vessels.
630	Retrolental fibroplasia
640	Detachment of retina

650 Tapetoretinal degeneration and allied conditions, including retinitis pigmentosa
660 Macular degeneration
670 Neoplasm of retina
680 Other affection of retina specified
690 Affection of retina, not specified

Optic nerve, optic pathway and cortical visual centers :

710 Optic nerve atrophy, optic neuritis or papilledema
730 Other affection of optic nerve, specified
740 Affection of optic nerve, not specified
750 Lesion of optic pathway or cortical visual centers

Vitreous (only to be used if classification elsewhere is impossible) :

810 Vitreous hemorrhage
880 Other affection of vitreous specified
890 Affection of vitreous not specified

Site and type indefinite or not reported :

940 Amblyopia ex anopsia
945 Amblyopia uni- or bilateral, not explained
950 Congenital nystagmus (only to be used if classification elsewhere is impossible)
980 Evidence insufficient for diagnosis
990 No report on site and type of affection.

INDEX OF DIAGNOSTIC TERMS INDICATING ETIOLOGY

	Code no.	
Accident, see trauma		
Act of violence (subdivide further by causal agent)	47.0	
Affection of central nervous system spec., not listed in class.	65.8	
Affection of central nervous system not spec.	65.9	
Agent causing burn, not caused by chemical or radiation (first classify by type of activity, external cause)	0.03	
Airgun (first classify by type of activity)	.07	
Albuminuria	63.0	
in pregnancy	66.0	
Alcohol, see methylalcohol	—	
Anemia	61.0	
Arachnoiditis optico-chiasmatica	65.8	
Arteritis temporalis	64.0	
Arteriosclerosis	64.0	
Auto-toxemia, auto-intoxication : focal sepsis (infection)	17.0	
non-infectious disease or disorder (classify by type of disease if known)	61.0	to 79.0
not specified	98.0	
Birth process	45.0	
Blood disease	61.0	
Blow	.09	

Brain :		
abscess of (classify by type of affection if known)	11.0	to 29.0
fever, see encephalitis, meningitis	—	
hemorrhage, cause not spec.	64.0	
injury at birth	45.0	
other injury of.. (classify by type of activity and causal agent)	40.0	to 49.0
tumor, neoplasm	65.2	
Brain tumor	65.2	
Bright's disease	63.0	
in pregnancy	66.0	
Brucellosis	26.0	
Burns (incl. chemical)	40.0	to 49.0
Cancer	78.0	
Carcinoma	78.0	
Causal agent of accident, poisoning or violence, specified, not listed in class	.18	
Causal agent of accident, poisonning or violence, not specified	.19	
Central nervous system :		
affection specified, not listed in class.	65.8	
affection not specified	65.9	
intracranial neoplasm	65.2	
multiple sclerosis	65.1	
Cerebro-vascular lesion	64.0	
Chemical causing burn	.01	
Childbirth disease or disorder affecting mother	66.0	
Complication of pregnancy	66.0	
Congenital (classify by cause if known)	81.0	to 89.0
Consanguinity	81.0	
Corpus alienum in eye	.10	
Dental disease	17.0	
Diabetes mellitus	62.0	
Dietary deficiency	67.0	
Dinitrophenol poisoning	.12	
Diphtheria	11.0	
Disorder of central nervous system :		
specified, not listed in class. not specified	65.8	
not specified	65.9	
Dysoxygenation	.15	
Encephalitis :		
(classify by primary etiology if known)		
epidemic	28.0	
lethargic	28.0	
type not specified	65.8	
Endocrine imbalance	78.0	
Erysipelas	28.0	
Evidence insufficient for diagnosis	98.0	
Explosive not caused by firearm or fireworks	.06	

Fall	.09
Firearm using explosive	.04
Fireworks	.05
Focal infection (sepsis)	17.0
Foreign body in eye	.10
General disease:	
specified, not listed in class.	78.0
not specified	79.0
Genetic origin:	
established	81.0
probable, presumed	82.0
Glandular deficiency	78.0
Gonorrhoea	12.0
Hemorrhage:	
brain (or intracranial) at birth	45.0
brain, following injury, see trauma	—
other vascular lesion	64.0
Hereditary origin:	
established	81.0
probable, presumed	82.0
Herpes zoster	28.0
High bloodpressure	64.0
Household activity	42.0
Hydrocephalus	65.8
Hypertension constitutional	64.0
Idiopathic	91.0
Infantile paralysis	28.0
Infectious disease specified, not listed in class.	28.0
Infectious disease, not specified	29.0
Influenza	28.0
Injury see trauma	—
Intracranial neoplasm	65.2
Kidney disease	63.0
in pregnancy	66.0
Lead	.13
Leprosy	27.0
Lesion of brain vessels	64.0
Leukemia	61.0
Lues, see Syphilis	—
Malformation	81.0 to 89.0
Malnutrition	67.0
Mastoiditis	17.0
Measles	13.0
Medical procedure (see also trauma)	46.0
Meningitis (cerebral, spinal or both) (classify by primary etiological factor if known)	
epidemic	14.0

meningococcal	14.0	
infectious type, not specified	29.0	
not specified	65.8	
Meningococcal infection	14.0	
Metastatic infection	17.0	
Methylalcohol	.11	
Military activity	41.0	
Multiple sclerosis	65.1	
Neisserian infection	12.0	
Neoplasm in cerebro	65.2	
Nephritis	63.0	
in pregnancy	66.0	
No report on etiology	99.0	
Non-infectious systemic disease :		
specified, not listed in class.	78.0	
not specified	79.0	
Nutritional deficiency	67.0	
Occupational activity	40.0	
Onchocerciasis	24.0	
Operation surgical (see also trauma)	46.0	
Oxycephaly	81.0	or 88.0
Pemphigus (general)	78.0	
if only eye attacked	91.0	
Pertussis	28.0	
Play or sport other than occupational	43.0	
Pneumococcic infection	28.0	
Poisoning	40.0	to 49.0
Poliomyelitis	28.0	
Pregnancy, complication of	66.0	
Prenatal influence :		
specified, not listed in class.	88.0	
not specified	89.0	
Purpura	61.0	
Purulent infection of eye, organism not specified	29.0	
Quinine	.14	
Radiation	.02	
Rheumatism	78.0	
Rheumatic fever	28.0	to 29.0
Rubella	23.0	
Sarcoidosis	68.0	
Scarlet fever	16.0	
Sclerosis multiplex	65.1	
Senility	91.0	
Septicemia	17.0	
Sharp or pointed object	.08	
Sinusitis	17.0	
Slingshot	.07	
Smallpox	18.0	
Sport or play other than occupational	43.0	

Staphylococcic infection	28.0	
Streptococcic infection	28.0	
Suppurative eye condition, cause not specified	29.0	
Surgical procedure (see also trauma)	46.0	
Syphilis :		
prenatal	19.1	
postnatal	19.2	
pre- or postnatal not specified	19.9	
Systemic disease :		
specified, not listed in class.	78.0	
not specified	79.0	
Tabes	19.1	to 19.9
Teeth	17.0	
Tobacco-poisoning	48.18	
Toxemia in :		
focal infection	17.0	
pregnancy	66.0	
other disease, classify by type of disease	—	
Toxoplasmosis		
Prenatal	25.1	
Postnatal	25.2	
Pre- or postnatal not specified	25.9	
Trachoma	20.0	
Traffic or transportation, other than occupational	44.0	
Trauma	40.0	to 49.0
act of violence	47.0	
activity specified, not listed in class.	48.0	
activity not specified	49.0	
at birth from any cause	45.0	
Brain :		
at birth	45.0	
other, classify by type of activity	40.0	to 49.0
game, any type, unless engaged in professionally	43.0	
horseplay	43.0	
household chores, repair, other home activities	42.0	
medical treatment :		
malpractice	46.0	
quackery	46.0	
unexpected harmful results of medical treatment for other parts of body	46.0	
military activity	41.0	
occupational activity	40.0	
operations or surgical treatment :		
postoperative complications of eye	—	

(classify by original cause)	
unsuccesful (operation classify by original cause)	—
unexpected harmful	
results of surgery in other parts of body	46.0
unwarranted operative interference	46.0
pedestrian travel	44.0
play	43.0
pranks	43.0
recreation any type	43.0
sport any type unless engaged in professionally	43.0
transportation any type	44.0
vehicle	44.0
war : combatant	41.0
non-combatant	41.0
Tuberculosis	21.0
Tumor :	
intracranial	65.2
carcinoma, causing metastatic tumors in eye	78.0
Typhoid fever	22.0
Unknown etiology :	
born blind	89.0
unknown to science	91.0
e.g. cataract senile type, glaucoma non congenitum, myopia, unless hereditary origin established	
evidence insufficient for diagnosis	98.0
no report on etiology	99.0
Uremia	63.0
in pregnancy	66.0
Varicella	28.0
Variola	18.0
Vascular disease	64.0
Violence, see act of violence	—
Vitamin deficiency	67.0
War injury :	
combantant)	
non-combantant)	41.0
Whooping cough	28.0

INDEX OF DIAGNOSTIC TERMS INDICATING SITE AND TYPE OF AFFECTION.

Ablatio retinae	640
Ablatio retinae falciformis	148
Achromatopsia totalis	680
Acute endophthalmitis	130

Affection specified, not listed in classification of:	
choroid	580
ciliary body	580
conjunctiva	280
cornea	380
eyeball	180
iris	580
lens	480
optic nerve	780
retina	680
uvea	580
vitreous	880
Affection not specified of:	
choroid	590
ciliary body	590
conjunctiva	290
cornea	390
eyeball	190
iris	590
lens	490
optic nerve	740
retina	690
uvea	590
vitreous	890
Albinism:	
generalized	141
ocular	141
Allied conditions of tapetoretinal degeneration	650
Amaurosis uni- or bilateral, not explained	945
Amaurosis cong. Leber	650
Amblyopia uni- or bilateral, not explained	945
Amblyopia ex anopsia	940
Angioid streaks	580
Aniridia	145
Anomalies:	
congen. multiple	147
congen. specified, not listed in class.	148
congen. not classified	149
Anophthalmos cong. at birth	142
Anophthalmos, surgical, acquired (classify by primary lesion if known)	150
Aphakia acquired	410
Aphakia congen.	148
Aplasia maculae	148
Arteriosclerotic disease of choroid	580
Atrophia gyrata choroid et retinae	580
Atrophic globe	150

Atrophy of :	
choroid after choroiditis	550
eyeball	150
optic nerve	710
retina after retinitis	610
retina after retinopathy	610
retina and choroid after retinochoroiditis	560
Avulsion of eyeball	180
Buphthalmos	143
Burns of (including chemical) :	
cornea	380
eyeball	180
Cataract :	
after	410
postoperative	410
primary	410
secondary = after	410
secondary = complicata (classify by primary lesion)	—
Cerebral lesion :	
see lesion of optic pathway and cortical visual centers	750
Chalcosis bulbi	180
Choked disc	710
Chorioretinitis :	
myopic	122
other	560
Choroid :	
affection specified, not listed in class.	580
affection not specified	590
arteriosclerotic disease of	580
atrophia gyrata	580
atrophy after choroiditis	550
choroideremia	580
coloboma of	146
melanoblastoma of (melanosarcoma)	590
Choroideremia	580
Choroiditis :	
myopic	122
other	550
Coats exsudative retinitis	610
Coloboma :	
at birth (any part)	146
surgical coloboma of iris : classify by primary lesion	—

Congenital anomaly:
  multiple 147
  specified, not listed in class. 148
  not specified 149
Congenital defect, see congenital anomaly —
Congenital hemeralopia 650
Congenital nystagmus 950
  (only to be used if class. elsewhere is impossible)

Conical cornea:
  acquired 370
  congenital (at birth) 148

Conjunctiva:
  affection of, specified, not listed in class. 280
  affection of, not specified 290

Conjunctivitis purulenta:
  of newborn 211
  other than newborn 212

Contusion of:
  cornea 380
  eyeball 180

Cornea:
  abscess of 313
  affection of, specified, not listed in class. 380
  affection, not specified 390
  burns of (including chemical) 380

  conical:
    congen. 148
    acquired 370
  contusion of 380

  degeneration of, dystrophy of } primary 320

  dystrophy due to physical agents 380
  injuries of 380
  hypopyon ulcer of 313
  leukoma, macula, opacities, scars, staphyloma } classify by primary affection if possible, otherwise 390

pannus trachomatosus	313
pannus eczematosus	312
ulcer of	313
xerosis	360
Corpora aliena in eye	180
Cortical visual centers:	
lesion of	750
Cyclitis	510
Defect, developmental or congenital, see congenital anomaly	—
Degeneratio hyaloidea-retinalis	680
Detachment of retina	640
Detachment of retina, cause myopia	121
Developmental defect, see congenit. anomaly	—
Dialysis retinae	640
Dislocated lens	420
Disorder of central artery or vein of retina	610
Disorganized eyeball	150
Dysgenesis mesodermalis corneae et iridis	148
Eales' disease	610
Embolism of central artery	610
Endophthalmitis	130
Enucleation (classify by primary lesion if known)	150
Evisceration (classify by primary lesion if known)	150
Excised eyeball (classify by primary lesion if known)	150
Exophthalmos	180
Eyeball:	
(see specific affection or anomaly if known)	
affection, specified, not listed in class.	180
affection not specified	190
atrophic	150
avulsion of	180
degenerative changes due to physical agents:	
specified, not listed in class.	180
not specified	190
disorganized	150
enucleated (classify by primary lesion if known)	150
eviscerated (classify by primary lesion if known)	150
phthisic (classify by primary lesion if known)	150
rudimentary (congenital)	142
Foreign bodies in:	
cornea	380
eye	180

Glaucoma :	
adult type	110
congenital	143
juvenile	110
infantile	143
secondary (classify by primary lesion)	—
Glioma retinae	670
Globe, see eyeball	—
Haemophthalmos	180
Hemeralopia congenital	650
Hemorrhage :	
intra-ocular (classify by primary lesion if known)	810
retinal	610
subhyaloid	610
Hole in macula :	
senile	660
traumatic	680
coloboma maculae congen.	146
Hyalitis (classify by primary lesion if possible)	880
Hydrophthalmos	143
Hypertension, see glaucoma	—
Hypopyon (keratitis, ulcer)	313
Hypopyon (iritis)	510
Infantile glaucoma	143
Interstitial keratitis	311
Intra-ocular foreign body	180
Irideremia	145
Irido-choroiditis	510
Iridocyclitis :	
sympathetic	570
other	510
Iris :	
affection specified, not listed in class.	580
affection not specified	590
coloboma of (congenital)	146
Iritis :	
sympathetic	570
other	510
Keratectasia (classify by primary lesion if known)	380
Keratitis :	
affection specified, not listed in class.	318
affection not specified	319
disciformis	318
eczematous	312

  interstitial 311
  parenchymatous 311
  Phlyctenular 312
  profunda 311
  sclerotic 314
  ulcerative 313
  xerotic 360
Keratoconjunctivitis phlyctenular 312

Keratoconus:
  congenital 148
  acquired 370
Keratoglobus with hypertension, see hydrophthalmos —
Kerato-iritis 530
Kerato-iridocyclitis 530
Keratomalacia 360

Lens:
  affection specified, not listed in class. 480
  affection not specified 490
  degeneration of 410
  dislocated 420
  ectopic 420
  luxation of 420
  opacity of 410
  subluxation of 420
Lesion of cortical visual centers 750
Lesion of optic pathway 750
Lesions (multiple) of eyeball after trauma 180
Lesion of retinal vessels 610
Leukoma corneae (classify by primary lesion if known) 390
Leukoma adhaerens (classify by primary lesion if known) 390
Luxatio lentis 420

Macula:
  corneal (classify by primary lesion if known) 390

  hole in the:
  senile 660
  traumatic 680

  degeneration of:
  senile 660
  juvenile (Stargardt) 660
Megalocornea with hypertension (see hydrophthalmos) —
Megalophthalmos 143

Melanoblastoma of :	
Choroid, ciliary body, iris	580
Microphthalmos	144
Multiple congen, anomalies	147
Myopia :	
malignant, progressive, with retinal detachment	121
malignant, progressive, detachment of retina not specified	122
Myopic choroiditis	122
Near-sightedness, see Myopia	—
Neoplasm :	
(classify by part of eye affected)	
choroid	580
cornea	380
iris	580
retina	670
optic nerve	730
intracranial, classify under etiology	—
Neuritis :	
intra-ocular	710
retrobulbar	710
of optic pathway	750
Neuroretinitis	710
Nystagmus congenital (only to be used if classification elsewhere is impossible)	950
Occlusion of central artery or vein	610
Ocular atrophy	150
Ocular avulsion	180
Ocular contusion	180
Opacities :	
corneal (classify by primary lesion if known)	390
lens	410
vitreous (only to be used if classification elsewhere is impossible)	880
Ophthalmia = Ophthalmitis :	
acute	130
purulenta neonatorum	211
purulenta other than newborn	212
scrophulous	312
sympathetic	570
Optic nerve :	
affection specified, not listed in classification	730
affection not specified	740
atrophy of, primary, postneuritis, post papillitic	710

Optic neuritis	710
Optic pathway lesion	750
Panophthalmitis	130
Papilloedema	710
Papillitis	710
Pemphigus conjunctivae	280
Perforating :	
Ulcer of cornea	313
wound of cornea	380
wound of eyeball	180
Perivasculitis retinae	610
Phlyctenular ulcer	312
Phthisis bulbi	150
Prenatal vascular anomalies	148
Pseudoglioma : (classify by primary lesion if possible)	—
otherwise	148
Pterygium	380
Pupil :	
occlusion/seclusion of	510
Purulent ophthalmia :	
of newborn	211
other than newborn	212
Retina :	
ablatio	640
ablatio falciformis	148
affection specified, not listed in class.	680
affection not specified	690
arteriosclerotic disease of	610
degeneration of	650
detached	640
dysplasia	148
embolism of artery	610
glioma of	670
occlusion of artery or vein	610
retinoblastoma	670
thrombosis of artery or vein	610
undeveloped	148
Retinal detachment	640
Retinal dysplasia	148
Retinitis :	
circinata	610
exsudativa Coats	610
hemorrhagic	610
pigmentosa	650
proliferans	610
punctata albescens	650

Retinoblastoma	670
Retinochoroiditis	560
Retinopathy	610
Retinoschizis	680
Retrobulbar :	
lesion of optic pathway	750
neuritis	710
Retrolental fibroplasia	630
Rudimentary eyeball (congen.)	142
Sarcoma of choroid	580
Sclerosing keratitis	314
Sclero-iridocyclitis	530
Siderosis bulbi	180
Staphyloma : (classify by primary lesion if known)	
otherwise	390
posterior	122
Striae angioides	580
Structural anomaly, see congen. anomaly	—
Surgical anophthalmos (only to be used if primary lesion is unknown)	150
Sympathetic :	
inflammation, iridocyclitis, opthalmitis, uveitis	570
Synchysis	880
Tapetochoroidal degeneration	580
Tapetoretinal degeneration	650
Thrombosis of central artery or vein	610
Trichiasis, classify by primary eye lesion	—
Tumor, see neoplasm	—
Ulcerative keratitis	313
Ulcus serpens	313
Uveitis :	
sympathetic	570
all other	510
Vasculitis retinae	610
Vitreous (only to be used if classification elsewhere is impossible)	
affection specified, not listed in class.	880
affection not specified	890
foreign body in	180
hemorrhage	810
Wound of : cornea	380
eyeball	180
Xerophthalmia	360
Xerosis	360

SOME EXAMPLES OF CLASSIFICATION OF CLINICAL ENTITIES AND DIAGNOSIS

Achromatopsia totalis	
genetic origin established	81.0/680
genetic origin not established	82.0/680
Albinism :	
genetic origin establised	81.0/141
genetic origin not established	82.0/141
Amaurosis cong. Leber :	
genetic origin established	81.0/650
genetic origin not established	82.0/650
Amaurosis e causa ignota	91.0 or 98.0/945
Amblyopia cause in cerebro not further specified	65.9/750
Amblyopia e causa ignota	91.0 or 98.0/945
Aniridia :	
genetic origin established	81.0/145
genetic origin not established	89.0/145
Blennorrhoea :	
neonatorum gonorrh.	12.0/211
adultorum gonorrh.	12.0/212
Buphthalmos :	
genetic origin established	81.0/143
genetic origin not established	89.0/143
Campeyes	67.0/710
Cataract :	
congenita	
hereditary origin established	81.0/410
cause rubella	23.0/410
cause other disease of mother in pregnancy specified, classify by this lesion	—
if unknown	89.0/410
senilis	91.0/410
Choroideremia :	
genetic origin established	81.0/580
genetic origin not established	82.0/580
Chorioretinitis centralis toxopl. :	
prenatal	25.1/560
postnatal	25.2/560
pre- or postnatal not specified	25.9/560

Coloboma :	
maculae verum	
genetic origin established	81.0/146
genetic origin not established	82.0/146
any other part, excl. surgical :	
genetic origin established	81.0/146
genetic origin not established	89.0/146
Congenital cataract, see cataract congenita	—
Crouzon's disease :	
genetic origin established	81.0/710
genetic origin not established	82.0/710
Diabetic retinopathy	62.0/610
Dysgenesis mesodermalis corneae et iridis (Rieger) :	
genetic origin established	81.0/148
genetic origin not established	82.0/148
Eales' disease :	
if cause general vascular disease	64.0/610
if cause tuberculosis	21.0/610
if cause unknown	91.0/610
Familiar corneal degenerations (Groenouw, Haab, Dimmer etc.)	81.0/320
Glaucoma (any type excluding infantile and secondary) :	
genetic origin established	81.0/110
genetic origin not established	91.0/110
Iridocyclitis, uveitis :	
cause rheuma	78.0/510
cause sarcoidosis	68.0/510
sympathetic : see sympathetic ophthalmia	—
syphilis postnatal	19.2/510
toxoplasmosis postnatal	25.2/510
tuberculosis	21.0/510
undertermined origin	91.0/510
Keratitis :	
e lagophthalmo :	
cause cicatricial (trauma)	40.0 to 49.0/318
exophthalmos (endocrin)	78.0/318
paresis N. VII	65.8/318
parenchymatosa heredoluetica	19.1/311
Keratitis trachomatosa	20.0/313
Keratoconjunctivitis rosacea	78.0/318
Leber's optic atrophy	81.0/710
Leber's amaurosis congenita, see amaurosis cong. Leber	

Macular aplasia :	
genetic origin established	81.0/148
genetic origin not established	82.0/148
Macular dystrophy (Stargardt) :	
genetic origin established	81.0/660
genetic origin not established	82.0/660
Microphthalmos :	
genetic origin established	81.0/144
genetic origin not established	82.0/144
Mooren's corneal ulcer	91.0/318
Myopic chorioretinal atrophy :	
genetic origin established	81.0/122
genetic origin not established	91.0/122
with retinal detachment :	
genetic origin established	81.0/121
genetic origin not established	91,0/121
Oguchi's disease :	
genetic origin established	81.0/650
genetic origin not established	82.0/650
Optic nerve atrophy :	
cause : birth process	45.0/710
brain tumor	65.2/710
hereditary :	
dominant	81.0/710
Leber's	81.0/710
hydrocephalos	65.8/710
nutritional deficiency (e.g. campeyes)	67.0/710
unknown to science	91.0/710
vascular disesase	64.0/710
Retinal detachment :	
cause : aphakia : classify by primary cause	
with myopia :	
genetic origin established	81.0/121
genetic origin not established	82.0/121
secondary to neoplasm, uveitis etc. classify by primary lesion	—
senile	91.0/640
traumatic, classifiy for etiology by type of activity and by causal agent	/640
e causa ignota	91.0/640

Retinoblastoma :	
genetic origin established	81.0/670
genetic origin not established	91.0/670
Retinoschizis :	
genetic origin established	81.0/680
genetic origin not established	82.0/680
Retrolental fibroplasia	46.15/630
Senile cataract	91.0/410
Senile macular degeneration	91.0/660
Striae angioides :	
genetic origin established	81.0/580
genetic origin not established	82.0/580
Sympathetic ophthalmia, classify by etiology of the injury of the first eye, by site and type	— /570
Taperoretinal degeneration, dystrophy, dysfunction (primary) also as part of syndrome (e.g. Biedl-Bardet, Spielmeyer Vogt etc.) :	
genetic origin established	81.0/650
genetic origin not established	82.0/650
Trachoma	20.0/313
Vascular retinopathy :	
cause general vascular disease	64.0/610
otherwise	91.0/610
Xerophthalmia	67.0/360

SOME EXAMPLES OF CLASSIFICATION OF SYNDROMES

Apert :	
genetic origin established	81.0/710
genetic origin not established	82.0/710
Bardet-Biedl :	
genetic origin established	81.0/650
genetic origin not established	82.0/650
Behr :	
genetic origin established	81.0/710
genetic origin not established	82.0/710

Grönblad-Strandberg :	
genetic origin established	81.0/580
genetic origin not established	82.0/580
Harada	91.0/510
v. Hippel-Lindau :	
genetic origin established	81.0/670
genetic origin not established	82.0/670
Hurler :	
genetic origin established	81.0/320
genetic origin not established	81.0/320
Marchesani, Marfan :	
genetic origin established	81.0/420
genetic origin not established	82.0/420
Refsum :	
genetic origin established	81.0/650
genetic origin not established	82.0/650
T. Sjögren :	
genetic origin established	81.0/410
genetic origin not established	82.0/410
Spielmeyer-Vogt :	
genetic origin established	81.0/650
genetic origin not established	82.0/650
Still	91.0/510
Usher :	
genetic origin established	81.0/650
genetic origin not established	82.0/650
Vogt-Koyanagi	91.0/510

CHAPTER TEN. Cross tabulations of causes of blindness, 1958 — 1962, by sex in age groups : the same for 1/8'66 — 1/8'67.

Table A1. 91 blind boys, under 17 years of age seen at Undaan 1958, visual acuity less than $1^1/_2/60$. Causes of blindness according to site/type and etiology.

	12	13	19 .9	20	65 .8	65 .9	67	89	98	99	Total
144								4			4 (1 cured)
150										3	3
212	2										2 (1 died)
313				3					:1		4 :1 died)
315									1	:12	13 :2 died)
316										1	1
360	:2	1			:1		:32 8†? 3†				47 (6 died, 2 died? later)
380.5										4	4
410								2			2 (1 died)
510			1						1		2
550									1		1
560										1	1
710			1		1	1			1		4
940								1			1
	4	1	2	3	2	1	43	7	5	21	90 + 1: 91

not included in above tabulation: 18+89/143-190.
Of 91 boys 1 was later found to be cured, 15 had died, 1 was heard to have died (results of home visits).
Summary: Eyeball: 7.7% ; Conjunctiva (Gonorrh.) 2.2%; Cornea: 74,4% (Xerophth. 47%)
Lens: 2.2% ; Uvea: 4.4% ; Optic nerve: 4.4% ; Other: 1.1%.

Table A2. 100 blind boys, under 17 years of age, seen at Undaan 1959. Visual acuity less than $1^1/_2/60$. Causes of blindness according to site/type and etiology.

	12	19 .1	61	65 .2	65 .8	67	81	89	91	98	99	Total
144								3				3
146								1				1
147								2				2
150					1†?						2	3
211	2											2
212	6											6
313	1											1
315										2	:8	10 (:1 died)
316											3	3
360						46 2† 5†?						53 (2 cured, 7 died)
380.5											2	2
410								5				5
510										1		1
550		1										1 (died)
560										1		1
670							1†					1 (died)
710		1	1	:1								3 (:1 died?)
940									1			1
990					1							1
	9	2	1	1	2	53	1	11	1	4	15	100.

Of 100 boys 2 were found cured, 9 had died, 1 was heard to have died.

Summary : Eyeball 9% ; Cunjunctiva (gonorrh.) : 9%; Cornea: 68% (Xerophth. 53%);
Lens: 5%; Uvea : 3% ; Retina : 1% ; Optic nerve : 3% ; Other : 5%.

Table A3. 77 blind boys, under 17 years of age, seen at Undaan 1960. Visual acuity less than $1^1/_2/60$. Causes of blindness by site/type and etiology.

	12	21 19.1	20	65 .2	65 .8	65 .9	67	68	89	98	99	Total
110									1			1 (cured)
143									1			1 (died)
146									1			1 (cured)
150			1								2	3
211	3											3 (1 cured)
313			1								1	2
315								:1	1	2	:6	10 (: 2 died)
360		1					37 3† ?					 41 (6 died)
380.5											2	2
410									5			5 (1 died, 1 died? later)
550									1			1 (died)
560									1			1
710				1†	2	1					:1	5 (: 1 died)
Total	3	1	2	1	2	1	40	1	11	2	13	77.

Of 77 boys 6 were found cured, 10 had died, 1 was heard to have died.
Summary : Eyeball : 7.8% ; Conjunctiva (gonorrh) : 3.9% ; Cornea : 71.4% (Xerophth. 53%) ;
Lens : 6.5% ; Uvea 3.9% ; Optic nerve : 6.5%.

Table A4. 74 blind boys, under 17 years of age, seen at Undaan, 1961. Visual acuity less than $1^1/_2/60$. Causes of blindness by site/type and etiology.

	12	65 .8	65 .9	67	81	89	91	98	99	Total
110							1			1
122						2				2
142						1				1
143					:2	1				3 (:1 died)
147						2				2
150									2	2
190						1				1 (died)
211	1									1
212	3									3
315								2	6	8
320						1				1
360		1		25 2† 3† ?						(2 cured) 31 (1 died)
380.5									2	2 (1 died)
410						8				8 (3 died)
560								1	:1	2 (: 1 cured)
710		:1	3			1				5 (: 1 died)
780						1				1
Total	4	2	3	30	2	18	1	3	11	74.

Of 74 boys 3 were found cured, 8 had died.
Summary: Eyeball: 16%; Conjunctiva (gonorrh.): 5.4%; Cornea: 67% (Xerophth. 42%) ;
Lens : 10.8% ; Uvea : 2.7% ; Optic nerve : 8%.

Table A5. 60 blind boys, under 17 years of age, 1962. Visual acuity less than $1^1/_2/60$. Causes of blindness by site/type and etiology.

	12	19 .1	20	21	43	65 .8	65 .9	67	89	98	99	Total
130											1	1
147								1	7			8 (1 died)
150											1	1
211	3											3
311		1										1
313			1	1							1	3
315						1† ?				1	5	7
360								13 1† 2† ?				(1 cured) 16 (1 died. 1 died?)
410									9			9 (2 cured, 1 died)
420									1			1
510										1		1
530										1		1
560		1										1
570					1							1
650									1			1
710						2† ?	1			1	1	5
Total	3	2	1	1	1	3	1	17	18	4	9	60

Of 60 boys 3 were found to be cured, 3 had died, 1 was heard to have died.

Summary : Eyeball : 16.6% ; Conjunctiva (gonorrh.) : 5% ; Cornea : 45% ; (Xerophth. 26.6%) ;
Lens : 16.6% ; Uvea : 6.6% ; Retina : 1.6% ; Optic nerve : 8.3%.

Table A6. 69 blind girls, under 17 years of age, 1958. Causes of blindness by site/type and etiology. Visual acuity less than $1^1/_2/60$.

	12	13	18	19 .9	65 .8	65 .9	67	89	91	98	99	Total
110									3			3
141								1				1 (cured)
144								1				1
147								1				1
150											2	2
212	7											7 (1 died)
313		1									1	2
315			1					1			9	11
360	1						22 1† 3† ?					27 (10 died, 1 died?)
380.5											5	5
410								3				3 (1 cured)
510				1								1
560										1		1
710					1						1	2
780					1	1						2
Total	8	1	1	1	2	1	26	7	3	1	18	69.

Of 69 girls 2 were found cured. 11 had died, 1 was heard to have died.

Summary : Eyeball : 11.6% ; Conjunctiva (gonorrh.) : 10% ; Cornea : 60% ; (Xerophth. 39%) ;
Lens : 4% ; Uvea : 2.9% ; Optic nerve : 5.8%.

Table A7. 66 Blind girls under 17 years of age, seen at Undaan 1959. Visual acuity less than $1^1/_2/60$. Causes of blindness by site/type and etiology.

	12	12 /89	13	18	20 .1	65 .8	67	89	91	98	99	Total	
143								2				2	
144		1										1	
147								1				1	
150				1							1	2	
212	3											3	
313			1		1							2	
315	2										5	7	
360	1						31 1† 1†?					34	(5 died, 1 died?)
380.5											1	1	
410								3				3	(1 died)
560										1		1	
670									1			1	(died)
710						3		1			3	7	
980											1	1	
Total	6	1	1	1	1	3	33	7	1	1	11	66.	

Of 66 girls 7 were found to have died, 1 was said to have died.

Summary : Eyeball : 9,9% ; Conjunctiva (gonorrh.) : 10.7% ; Cornea : 66.6% (Xerophth. 51.5%)
Lens : 4.9% ; Uvea : 1.8% ; Retina : 1.8% ; Optic nerve : 16% ; Other : 1.8%.

Table A8. 49 blind girls, under 17 years of age, seen at Undaan 1960. Causes of blindness by site/type and etiology. Visual acuity : less than $1^1/_2/60$.

	12	12 /19.1	19 .1	19 .9	22	65 .9	67	89	91	98	99	Total
110								2				2
144								2				2
147								1				1
148								1				1 (died)
150											1	1
211		1										1
212	3											3 (1 died)
311			1									1
315				1				1			3	5
318				1								1
360							13 1† 1†?					(1 cured) 15
380.5											2	2
410								4				4 (1 died)
510										1		1
530											1	1
560			:1							2		3 (: 1 died)
670									1			1
710					1	1		2				4 (1 died)
Total	3	1	2	2	1	1	15	13	1	3	7	49.

Of 49 girls 1 was found cured, 5 had died, 1 was heard to have died.

Summary : Eyeball : 14% ; Conjunctiva (gonorrh.) : 8% ; Cornea: 49% (Xerophth. 30.6%);
Lens : 16% ; Uvea : 10.2% ; Retina : 2%, Optic nerve : 8%.

Table A9. 56 blind girls, under 17 years of age, seen at Undaan 1961. Visual acutiy; less than $1^1/_2/60$. Causes of blindness by site/type and etiology.

	12	12/20	13	18	20	65.9	67/19	67	81	82	89	98	99	Total
143											1			1
144											1			1
150				1									2	3
212	1	1												2
313					1							1	2	4
315			1								2		6	9
316													1	1
360							1	17 2† 3†?						(1 cured) 23 (1 died)
380.5													1	1
410									1		:4			5 (: 1 died)
550												1		1 (cured)
610											1			1 (Coats?)
650										1				1
710						2			x1					3 (x Crouzon)
Total	1	1	1	1	1	2	1	22	2	1	9	2	12	56.

Of 56 girls 2 were cured. 2 were found to have died.

Summary : Eyeball : 8.9% ; Conjunctiva (Gonorrh.) : 3.5% ; Cornea : 67% (Xerophth. 41%) ;
Lens : 8.9% ; Uvea : 1.7% ; Retina : 3.4% ; Optic nerve : 5.1%.

Table A 10. 58 blind girls, under 17 years of age, seen at Undaan 1962. Visual acuity less than $1^1/_2/60$. Causes of blindness by site/type and etiology.

	12	13	18	19.1	21	29	65 82	65 83	65.9	67	81	89	91	99	Total
142												1			1
144												1			1
147												4			4 (1 died)
150														1	1
190												1			1
212	1														1
313					1									1	2
315		1	1									1		4	7 (2 cured)
360										14 2†?					(1 cured) 16 (2 died)
380.5														2	2
410						1					1	7			9 (1 cured)
550							1								1
560				1								2			3
660									1						1
670													3		3 (died)
710					1			1				1		1	4
980														1	1 (hysterics)
Total	1	1	1	1	2	1	1	1	1	16	1	19	3	9	58.

Of 58 girls 5 were found cured, 6 had died.

Summary : Eyeball : 13.8% ; Conjunctiva (gonorrh.) : 1.7% ; Cornea : 46.5% (Xerophth. 27.6%)
Lens: 15%; Uvea: 6.9%; Retina: 6.9%; Optic nerve: 6.9%; Other: 1.7%.

Table A11. 59 blind boys, under 17 years of age, seen at Undaan 1/8'66—1/8'67. Visual acuity less than $1^1/_2/60$. Causes of blindness by site/type and etiology.

	12 /67	18	45	65	67	67 /98	89	89 /98	98	99	Total
110								1			1
130					1						1
147							3				3
150					2	1					3
311									1		1
315	1	2			7						10
316					1						1
360					19						19
380.5					7						7
410							5				5
510										1	1
560								1			1
610										1	1
650								1			1
710				1							1
990			1	1			1				3
Total	1	2	1	2	37	1	9	3	1	2	59

Summary:

Site/type.		
Eyeball	8	13.5%
Conjunctiva	—	—
Cornea	38	64.4%
Lens	5	9%
Uvea	2	3.4%
Retina	2	3.4%
Optic nerve	1	1.7%
Other	3	5%
Etiology.		
Gonorrhoea	1	1.7%
Syphilis	—	—
Trauma	1	1.7%
Neurol. disease	2	3.4%
Keratomalacia	38	64.4%
Congenital	12	20.3%
Ignota, Wr-	1	1.7%
Ignota	2	3.4%

Table A12. 61 blind girls, under 17 years of age, seen at Undaan 1/8'66—1/8'67. Visual acuity less than $1^1/_2/60$. Causes of blindness by site/type and etiology.

	12	18	20	65	65 .8	67	89	89 /98	91	98	99	Total
110							2	1				3
147							6					6
150	1	1				1					2	5
313							1					1
315			1			6	1					8
360						20						20
380.5	1					3					1	5
410							1					1
560										1		1
640									1			1
670									1			1
710				3						2		5
990				2	1						1	4
Total	2	1	1	5	1	30	11	1	2	3	4	61

Summary:

Site/type.		
Eyeball	12	21%
Conjunctiva	2	3.2%
Cornea	33	54%
Lens	1	1.6%
Uvea	1	1.6%
Retina	2	3.2%
Optivc nerve	5	8.2%
Other	4	6.5%
Etiology.		
Gonorrhoea	2	3.2%
Trachoma	1	1.6%
Neurol. disease	6	9.8%
Keratomalacia	30	49.1%
Congenital	12	19.6%
Ignota Wr-	3	4.9%
Ignota	4	6.4%

Table B 1. 387 blind boys, under 17 years of age, seen at Undaan 1958-62. Visual acuity : less than $1^1/_2/60$. Causes of blindness by site/type and etiology. Compilation of Tables A 1-5. Boys who were found on home visit to have been cured are left out.

	12	13	19 .1	19 .9	19 /21	20	21	43	61	65 .2	65 .8	65 .9	67	81	89	91	98	99	Total
110																1			1
122															2				2
130																		1	1
142															1				1
143														2	4				6
144															6				6
146															1				1
147													1		11				12
150						1					1							10	12
190															1				1
211	8																		8
212	11																		11
311			1																1
313	1					5	1										1	2	10
315											1				1		8	36	46
316																		4	4
320															1				1
360	2	1			1								175						179
380.5											2							10	12
410															27				27
510				1											1		3		5
530																	1	1	2
550			1												1		1		3
560			1												1		2	1	5
570								1											1
650															1				1
670														1					1
710			1	1					1	2	6	6			1		3	1	22
780															1				1
940															1	1			2
990											1								1
Total	22	1	4	2	1	6	1	1	1	2	11	6	176	3	62	2	19	66	386 + 1 : 387. + 89/143-18/190.

Summary.

Site/Type		
Eyeball	43	11%
Conjunctiva	19	4.9%
Cornea	253	68.4%
Lens	27	7%
Uvea	16	4.1%
Retina	2	less than 1%
Optic nerve	23	6 %
Other	3	less than 1%

Etiology		
Gonorrhoea	22	5.7%
Syphilis	6	1.5%
Trachoma	6	1.5%
Neurolog. disease	19	4.9%
Keratomalacia	176	45.3%
Congenital	65	16.8%
Ignota, Wr.-	19	4.9%
Ignota	66	16.8%

Table B2. 288 blind girls, under 17 years of age, seen at Undaan 1958-62. Visual acuity less than $1^1/_2/60$. Causes of blindness by site/type and etiology. Compilation of Tables A6-10. Girls who on home visit were found to be cured are left out.

	12	13	18	19	20	21	22	29	65 .8	65 .9	67	81	82	89	91	98	99	Total
110														2	3			5
142														1				1
143														4				4
144	1													5				6
147														7				7
148														1				1
150			2														7	9
190														1				1
211	1																	1
212	16																	16
311				1														1
313		2			2	1										1	4	10
315	2	2	2	1										5			25	37
316																	1	1
318				1														1
360	2										109							111
380.5																	11	11
410								1				2		19				22
510				1												1		2
530																	1	1
550									1									1
560				2										2		4		8
610														1				1
650													1					1
670															5			5
710						1	1		5	3		1		4			5	20
780									2									2
980																	2	2
Total	22	4	4	6	2	2	1	1	8	3	109	3	1	52	8	6	56	288.

Summary. Site/type.

Eyeball	34	11.8%
Conjunctiva	17	5.9%
Cornea	172	63%
Lens	22	7.6%
Uvea	12	4%
Retina	7	2.4%
Optic nerve	22	7,6%
Other	2	less than 1%.

Etiology.

Gonorrhoea	22	7.6%
Syphilis	6	2%
Trachoma	2	1%
Neurol. disease	11	3.8%
Keratomalacia	109	37.8%
Congenital	56	19%
Ignota, Wr-	6	2%
Ignota	56	19%

Table A 13. 284 blind men, age 16—41 years, seen at Undaan 1958. Visual acuity less than $1^1/_2/60$. Causes of blindness by site/type and etiology.

	12	12 /19	19 .9	20	20 /98	20 .1	28 01	28 03	44 09	44 10	65 .8	81	82	89	91	98	99	Total
110			2												10			12 + 91/110-313.
122															1	1		2
130			1													1	3	5
147												1						1
148														1				1
150									1	1							14	16
212	29																	29
311						1										2		3
313				22	1	2		1								5	9	40
315		1	6	2												2	82	93
316																	7	7
320																1		1
370.1				1													1	2
380							1											1
380.4																	2	2
380.5				1													5	6
410			1											1	1	1		4
420																1		1
510			6	1												1	1	9
530			1													1	1	3
550																5		5
560			6													2		8
650													3					3
660																1	1	2
710			15								2			1		6	1	25
810																2		2
Total	29	1	38	27	1	3	1	1	1	1	2	1	3	3	12	32	127	283 + 1: 284.

Different cause : 5 × "last eye". Different site : 40 × "last eye", 26 × right eye.

Table A 14. 246 blind men, age 16-41 years, seen at Undaan 1959. Visual acuity less than $1^1/_2/60$. Causes of blindness by site/type and etiology.

	12	12 /19	18	19 .9	20	20 .1	27	28 01	28 04	49 08	65 /82	82	89	91	98	99	Total	
110														10			10	+ 20/510-315
130				2												2	4	98/315-510
150	3			1											1	16	21	98/510-315
211	1																1	99/315-530.
212	18	1															19	
311				1	1												2	65.2/710-98/315
313	5	1		1	17	3	1	1		1					1	4	35	99/315-91/410
315	6		2	4	3				1						3	63	82	99/510-91/420
316	4							1								7	12	99/510-91/110
320				1													1	
380.3											1				1		2	
380.4				1												1	2	
380.5	1															1	2	
410				1													1	
420				1									1				2	
510				4											10	1	15	
530				2												2	4	
550				1											1		2	
560				3											1		4	
650												1					1	
710				12							1				1		14	
810				1											1		2	
Total	38	2	2	36	21	3	1	2	1	1	2	1	1	10	20	97	238 +	8: 246.

Different cause: 7 × "last eye".
Different site: 40 × " last eye", 10 × right eye.

Table A 15. 214 blind men, age 16-41 years, seen at Undaan 1960. Visual acuity less than $1^1/_2/60$. Causes of blindness by site/type and etiology.

	12	12 /19	17	18	19 .9	20	20 .1	21	28 01	28 04	65 .7	65 /81	81	82	89	91	98	99		Total
110																9			9	+ 20/313-510
130									1			1					1	2	5	: 82/410-420
144															1				1	98/315-510
147															1				1	99/530-315
150						1				1					1			10	13	19.9/316-510
190																		1	1	: Marfan.
212	21																		21	
313				1	1	15				1							3	3	24	
315	1			1	3	3			2	1							6	40	57	12/212-18/150
316				1													5	3	9	91/410-19.9/510
320																	2		2	91/410-99/530
370.1							1											2	3	20/313-65.8/313
380.4						1												2	3	
380.5																	1	2	3	
390						2													2	
410					1								1			1			3	
420															1				1	
510					5												4		9	
530		1			1												1		3	
550					2														2	
560					3												2	1	6	
650														1	2				3	
710			1		14						1						5		21	
810								1									1	1	3	
Total	22	1	1	3	30	22	1	1	3	3	1	1	1	1	6	10	31	67	205 +	9 : 214.

Different cause : 3 × "last eye"
Different site : 28 × "last eye"
11 × right eye.

Table A16. 205 blind men, age 16-41 years, seen at Undaan 1961. Visual acuity less than $1^1/_2/60$. Causes of blindness by site/type and etiology.

	12	19 .9	19 /21	20	20 .1	21	45 .8	82	89	91	98	99	Total	
														+
110		1								13			14	19.9/315-530.
130												2	2	91/410-420
146									1				1	98/315-510(2 ×)
150												5	5	98/510-315
212	26												26	98/510-316
311				1									1	98/710-315
313		2		18	5						3	3	31	20.1/313-41/110
315				4	1		1				3	33	42	91/110-99/315
316												6	6	91/110-99/380.4.
320											1		1	
370											1		1	different cause :
370.1				1									1	7 ×" last eye"
380.4												3	3	
380.5												6	6	different site :
510		4									2		6	19 ×" last
530											1	1	2	eye"
560		6									5		11	7 × right eye
610			1			1							2	
640												1	1	
650								7					7	
710		17									4	3	24	
750												1	1	
810											1		1	
Total	26	30	1	24	6	1	1	7	1	13	21	64	195	+ 10: 205.

Table A 17. 190 blind men, age 16-41 years, seen at Undaan 1962. Visual acuity less than $1^1/_2/60$. Causes of blindness by site/type and etiology.

	12	18	19 .9	20	20 .1	28 01	28 04	40 09	46	62	89	91	98	99	Total	
																+
110												8			8	19.9/315-510
122													1		1	19.9/560-313
130													1		1	20/510-380.4
147											2				2	40.08/150-570
150	4													3	7	40.08/315-570
212	13														13	99/530-315
280			1												1	
313	2		2	1			1						3	4	23	19.9/313-89/144
315	5		1	1	1	1			1				8	29	47	40.08/313-99/150
316	1												2	5	8	40.08/420-19.9/410
370.1				1											1	89/143-91/510
380.4														3	3	99/315-40.08/313
380.5														5	5	99/380.5-91/410
410										1	1	6			8	99/380.5-91/110
510			3										7		10	
530			1												1	
550		1	1												2	
560			6								2		1		9	
610													1		1	
710			16					1					8		25	
990														1	1	
Total	25	1	31	13	1	1	1	1	1	1	5	14	32	50	177	+ 13 : 190.

Different causes: 3 × " last eye", 2 × right eye.
Different site: 26 × " last eye", 23 × right eye.

Table A 18. 106 blind men, age 16-41 years, seen at Undaan 1/8'66-1/8'67. Visual acuity less than $1^1/_2/60$. Causes of blindness by site/type and etiology.

	12	12?	19	20	20 /98	27	40	46	62	65 .8	81	81 /98	89	91	91 /98	98	99	Total
110				1							1			3	1			6
122														1	1			2
130							1											1
143													1					1
147													1					1
150	1						1										2	4
212	6																	6
313	1			4				1								1	2	9
315	1	2	1		1												16	21
316	4	1																5
380.4																	1	1
380.5	1																1	2
390						1												1
410								1	1					2	3			7
420														1				1
510															1	2		3
560			1													4	1	6
570							1											1
640														1		1		2
650												1						1
660			1															1
710			14							1						6		21
810														1		1		2
990																	1	1
Total	14	3	17	5	1	1	3	2	1	1	1	1	2	9	6	14	24	106

(2 persons from the lists 1958-62 came again during this year).

Table B 3. 1139 blind men, age 16-41 years, seen at Undaan 1958-62. Visual acuity less than $1^1/_2/60$. Causes of blindness by site/type and etiology. Compilation of tables A 13-17.

	12	17	18	19	20	21	27	28	40	44	45	46	49	62	65	81	82	89	91	98	99	Total
110				3															50			53
122																			1	2		3
130				3				1							1					3	8	16
144																		1				1
146																		1				1
147																1		3				4
148																		1				1
150	7			1	1			1		2								1		1	45	59
190																					1	1
211	1																					1
212	108																					108
280				1																		1
311				1	3																	4
313	8		1	6	94		1	3					1							15	23	152
315	13		3	14	15			5			1	1								22	247	321
316	5		1					1												7	28	42
320				1																4		5
370					4																3	7
380	1			1	3			1							1					2	30	39
410				3										1		1		2	8	1		16
420				1														2		1		4
510				22	1															24	2	49
530	2			5																3	4	14
550			1	4																6		11
560				24														2		11	1	38
610				1		1														1		3
640																					1	1
650																	12	2				14
660																				1	1	2
710		1		84					1				1		3			1		24	4	119
750																					1	1
810						1														4	1	6
990																					1	1
Total	145	1	6	175	121	2	1	12	1	2	1	1	2	1	5	2	12	16	59	132	410	1098

+ 41 : 1139

If the right eye's classification of 41 persons not included in the above crosstabulation (see Tables A 13-17) are taken as the decisive one, the following **summary** of causes of blindness in men of age 16-41 years can be given :

Site : Eyeball 139 + 4 :	143	12.8%
Conjunctiva 110 + 1 :	111	9.7%
Cornea 570 + 17 :	587	51.5%
Lens 20 + 5 :	25	2.1%
Uvea 112 + 12 :	124	10.8%
Retina 20	20	1.7%
Optic nerve 120 + 2	122	10.4%
Other 7	7	less than 1%

Etiology. Gonorrhoea 145 + 1 :	146	12.8%
Syphilis 175 + 5 :	180	15.9%
Trachoma 121 + 4 :	125	10.9%
Trauma 7 + 4 :	11	1.5%
Congenital 30 + 2 :	32	2.8%
Glaucoma 50 + 3 :	53	4.6%
Senile cataract 20 + 3 :	23	2%
Ignota, Wr — 132 + 9 :	141	12.7%
Ignota 401 + 9 :	410	36%.

Summary of Table A 18, 106 blind men, age 16-41 years, 1/8'66-1/8'67, visual acuity less than $1^{1}/_{2}/60$:

Site. Eyeball	15	14 %
Conjunctiva	6	5.6%
Cornea	39	36.7%
Lens	8	7.4%
Uvea	10	9.6%
Retina	4	3.7%
Optic nerve	21	18.8%
Other	3	2.8%

Etiology/Gonorrhoea	17	16%
Syphilis	17	16%
Trachoma	6	5.6%
Congenital	4	3.7%
Glaucoma	6	5.6%
Senile cataract	7	6.5%
Ignota, Wr —	15	14%
Ignota	14	13%

Table A 19. 232 blind women, age 16 - 41 years, seen at Undaan 1958. Visual acuity less than $1^1/_2/60$. Causes of blindness by site/type and etiology.

	12	12/19	17	19.9	20	20.1	20/91	28 03	28 04	46	49 08	64/98	65	66/67	81	82	91	98	99	Total	
110																	19			19	+ 19.9/110-610
130	1										1							1	1	4	19.9/810-510
150																			9	9	98/315-510(2×)
212	23	1																		24	89/146-98/510
311				1	1	1														3	99/315-91/110(2×)
313				1	27	5		2										3	5	43	
315					1	2				1								2	42	48	
316				2	1													1	11	15	
360									1					1						2	
370.1					1	1												1		3	
380.4					1														3	4	
380.5						1													11	12	
410							1													1	
420																	1			1	
510			1	2														2	3	8	
530																			1	1	
560				1		1												2	1	5	
610																			1	1	
650																1				1	
660															2					2	
680																		1		1	
710				9								1						4	1	15	
750																		1		1	
810				1																1	
950													1							1	
Total	24	1	1	17	32	11	1	2	1	1	1	1	1	1	2	1	20	18	89	225 + 7 : 232.	

Different cause : 5×"last eye", 2× right eye.
Different site : 19×"last eye", 20× right eye.

Table A 20. 231 blind women, age 16 - 41 years, seen at Undaan 1959. Visual acuity less than $1^1/_2/60$. Causes of blindness by site/type and etiology.

	12	12 /19	18	19 .9	20	20 .1	28 04	29	42	62 /64	65 .2	66	82	89	91	98	99	Total	
																			+
110	2			1											17			20	98/315-510
130	1			2	1											2	2	8	99/311-480
150					1												4	5	99/530-315
190																	1	1	
212	13	4																17	12/212-91/110
280					1													1	19.9/313-89/190
313	3			1	22	3						1				2	1	33	
315	9			1	4		1									4	50	69	42.09/313-12/212
316	4	1					1									2	5	13	
370.1	1				3	1												5	91/110-99/530
380.3																1		1	98/510-20/313
380.4					1												1	2	99/315-91/110 (2×)
380.5					1												2	3	
410									1					1				2	
410/640																1		1	
510		1		5												7	1	14	
530				1				1								1	2	5	
550																1		1	
560				2												2		4	
610										1								1	
650													1	2				3	
710			1	8							1					1	1	12	
Total	33	6	1	21	34	4	2	1	1	1	1	1	1	3	17	24	70	221 + 10 :	231.

Different cause: 3× "last eye".
Different site: 39× "last eye", 1× right eye.

Table A 21. 223 blind women, age 16-41 years, seen at Undaan 1960. Visual acuity less than $1^1/_2/60$. Causes of blindness by site/type and etiology.

	12	12 /19	18	19 .9	20	20 .1	21	28 04	29	49	61 /63	62	65 .9	67 /12	89	91	98	99	Total	
110					2											24	1	1	27	+ 19.9/510-315
130																	2	1	3	19.9/710-315
150					4												1	7	12	98/510-315
212	28	2																	30	
280				1															1	
311					1														1	
313				2	29	1		1	1								3	7	44	91/110-99/315 (2×)
315				4													4	26	36	99/530-12/212
316				2													3	4	9	99/510-91/110.
320																	1		1	
360														1					1	
370.1						1													1	
380.4					1														1	
380.5					2						1							4	7	
410					1							1				2			4	
510				2			1										9	1	13	
530				2													1	1	4	
560				1											1		1		3	
710			1	8						1			2				5	1	18	
Total	29	2	1	22	40	2	1	1	1	1	1	1	2	1	1	26	31	53	216 + 7: 223.	

Different cause : 10×"last eye"
Different site: : 40×"last eye", 8× right eye.

Table A 22. 218 blind women, age 16-41 years, seen at Undaan 1961. Visual acuity less than $1^1/_2/60$. Causes of blindness by site/type and etiology.

	12	12/19	19.9	20	20.1	45/61	65.2	66/67	81	89	91	98	99	Total	+
110			1								18			19	19.9/315-510
130												3	3	6	62/410-640
142										1				1	89/144-147
144										1				1	98/510-313
150	1												3	4	98/510-315
190							1							1	98/550-640
212	24	1												25	98/610-313
313			1	26	4			2				3	3	39	98/710-311
315	2	1	2	3								7	30	45	99/315-510
316			1									1	2	4	91/420-410
320			1											1	99/510-315
360								1						1	
370												1		1	49/150-99/315
370.1			1	3	1									5	91/110-98/380.5
380.4				1									1	2	91/110-99/316
380.5	1			1									3	5	91/410-20/315
410											2			2	98/480-20/313
510			4		1							4		9	
530			1									3		4	
560			3									2		5	
610												2		2	
650									5					5	
710			10			1						2		13	
750												1		1	
950										1				1	
Total	28	2	25	34	6	1	1	3	5	3	20	29	45	202 + 16:	218.

Different causes: 5 × "last eye", 1 × right eye.
Different site : 23 × "last eye", 17 × right eye.

Table A 23. 187 blind women, age 16-41 years, seen at Undaan 1962. Visual acuity less than $1^1/_2/60$. Causes of blindness by site/type and etiology.

	12	12 /19	18	19 .9	20	20 .1	20 /67	62	78	82	89	91	98	99	Total	
110												23			23	+ 89/142-313
122												1			1	89/142-144
130													1		1	19.9/380.5-530
150	2													10	12	20.1/370.1-530
212	13	1													14	91/410-316
311													1		1	98/530-315
313				1	16	5	1						2	1	26	98/530-316
315	3	1	1	4	4							1	8	22	44	20.1/370.1-42.8-313
316	2			1									1	2	6	89/142-19.9/313
318									1						1	98/315-91/410
320													1		1	98/313-91/110 (2x).
370.1													1		1	
380.4					1										1	
380.5														11	11	
410					1	1		1				5			8	
420											1				1	
510				2									2		4	
530			1										1		2	
550													2		2	
560				1											1	
650										1					1	
710				10									3		13	
Total	20	2	2	19	22	6	1	1	1	1	1	30	23	46	175 + 12 : 187.	

Different cause : 4 × "last eye", 2 × right eye.
Different site : 25 × "last eye", 19 × right eye.

Table A 24. 111 blind women, age 16-41 years, seen at Undaan 1/8'66-1/8'67. Visual acuity less than $1^1/_2/60$. Causes of blindness by site/type and etiology.

	12	12?	19	20	20 /98	40	81	89 /98	91	98	99	Total
110							1	1	23			25
122									3			3
130		1										1
150		2									3	5
212	2											2
313				5						2	6	13
315	6	2		1	1						10	20
316	1	2										3
370.1	1				1						2	4
380.5	1										2	3
410			1						2			3
510										2	1	3
560			1							1		2
570						1						1
650								1				1
710			4							11	3	18
810			1									1
990			1								1	2
Total	11	7	8	6	2	1	1	2	28	16	28	110 + 1: 111

+ 91/110-410, 610.

(1 blind person already in the lists 1958-62, came back in this year).

Table B 4. 1091 blind women, age 16-41 years, seen at Undaan 1958-62. Visual acuity less than $1^1/_2/60$. Causes of blindness by site/type and etiology. Compilation of Tables A 19-23.

	12	17	18	19	20	21	28	29	40	61	62	64	65	66	67	78	81	82	89	91	98	99	Total
110	2			2	2															101	1		108
122																				1			1
130	2			2	1				1												9	7	22
142																			1				1
144																			1				1
150	3				5																1	33	42
190													1									1	2
212	110																						110
280				1	1																		2
311				1	3																1		5
313	3			6	139		3	1						1	2						13	17	185
315	17		1	11	14		1		1											1	25	171	242
316	7			6	1		1														8	24	47
318																1							1
320				1																	2		3
360							1								3								4
370	1			1	11																3		16
380	1				10					1												36	48
410					4				1		2								1	7	2		17
420																			1	3			4
510	1	1		15	1	1															24	5	48
530			1	4				1													6	4	16
550																					3		3
560				8	1														1		7	1	18
610											1										2	1	4
650																	1	7	2				10
660																	2						2
680																					1		1
710			2	45					2			1	3								15	3	71
750																					2		2
810				1																			1
950													1						1				2
Total	147	1	4	104	193	1	6	2	5	1	3	1	5	1	5	1	3	7	8	113	125	303	1039 + 52 : 1091.

If the right eye's classification of 52 persons not included in the above tabulation (see Tables A 19-23) is taken as the decisive one, the following **summary** of causes of blindness in women 16—41 years old can be given:

Site: Eyeball	177 + 12:	189	17.3%	Etiology: Gonorrhoea	147 + 1:	148	13.5%
Conjunctiva	112 + 1:	112	10.3%	Syphilis	104 + 7:	111	10 %
Cornea	551 + 15:	566	51.9%	Trachoma	193 + 2:	195	17.8%
Lens	21 + 5:	26	2.4%	Trauma	5 + 2:	7	1 %
Uvea	85 + 12:	97	8.8%	Senile cataract	17 + 5:	22	2 %
Retina	17 + 1:	18	1.6%	Glaucoma	113 + 6:	119	10.8%
Optic nerve	73 + 2:	75	6.8%	Congenital	18 + 5:	23	2.1%
Other	3 + 1:	4	1%	Ignota, Wr —	125 + 15:	140	12.8%
				Ignota	303 + 10:	313	18.6%

Summary of causes of blindness of III women seen at Undaan 1/8'66-1/8'67 (Table A **24**)

Site. Eyeball	34	30.5%	Etiology. Gonorrhoea	18	16.2%
Conjunctiva	2	1.8%	Syphilis	8	7.2%
Cornea	43	38.7%	Trachoma	8	7.2%
Lens	3	2.7%	Trauma	1	less than 1%
Uvea	6	5.4%	Senile cataract	3	2.7%
Retina	1	less than 1%	Glaucoma 25 + 1:	26	23.4%
Optic nerve	18	16.2%	Congenital	3	2.7%
Other	3	2.7%	Ignota, Wr —	16	14.4%
			Ignota	28	25.2%

Table A 25. 178 blind men, over 40 years of age, seen at Undaan 1958. Visual acuity less than $1^1/_2/60$. Causes of blindness by site/type and etiology.

	12	19.9	20	20/91	20.1	27	81	82	91	98	99	Total	+
110				1					24			25	99/510—315
122										1		1	
130											1	1	91/410—99/311
150											3	3	99/315—91/110
190											1	1	
212	5											5	
311			1							1	1	3	
313			29		4	1				1	12	47	
315		2	1		1					2	23	29	
316											2	2	
370.1										1		1	
380.3											1	1	
380.4			3								4	7	
410		1		2					3	3		9	
510		7			1					2	1	11	
530											1	1	
550		3								1		4	
560		3										3	
580										1		1	
610										2		2	
640										1		1	
650							1	2				3	
710		10			1					2		13	
990											1	1	
	5	26	34	3	7	1	1	2	27	18	51	175	+ 3 : 178.

Different site : 13× "last eye", 18× right eye.

Table A 26. 159 men, over 40 years of age, seen at Undaan 1959. Visual acuity less than $1^1/_2/60$. Causes of blindness by site/type and etiology.

	12	12 /19	18	19 .9	20	20 .1	28 04	62 /63	65 .8	82	91	98	99	Total	+
110											21			21	19.9/315-510
130													1	1	19.9/510-315 (2x)
150													5	5	98/315-510
212	2	1												3	19.9/510-89/146
311					1									1	91/110-99/315
313	1				27	1	1					1	5	36	91/110-99/ 380.4 (2x)
315	1		1		3							1	26	32	
316					1									1	
319				1										1	
370.1					1									1	
380.2													1	1	
380.4					2								3	5	
380.5													2	2	
410				2	1	1					2			6	
420												1		1	
510				5								3	1	9	
550				4								1		5	
560				5					1			2		8	
590												1		1	
610								1						1	
650										2				2	
710				8										8	
Total	4	1	1	23	36	2	1	1	1	2	23	10	44	151 + 8: 159.	

Different causes : 9× "last eye".
Different site : 9× "last eye", 8 × right eye.

Table A 27. 189 blind men, over 40 years of age, seen at Undaan 1960. Visual acuity less than $1^1/_2/60$. Causes of blindness by site/type and etiology.

	12	18 /19	19 .9	20	20 .1	49	62	65 .2	82	91	98	99	Total	
110			1							36			37	+ 98/313-510
130				1									1	98/510-315
150						1						4	5	91/110-315
212	3												3	
311			1								1		2	91/410-98/313
313				43	1	1					1	4	50	99/315-91/110
315		1	1					1			3	16	22	
316			2									2	4	
370			1										1	
370.1				1	1								2	
380.4				1							1	3	5	
380.5												1	1	
410			2	3			1			4			10	
420			1										1	
510			3								8	1	12	
560			1										1	
610			2								2		4	
640											1		1	
650									3				3	
660												1	1	
710			12								4	1	17	
Total	3	1	27	49	2	2	1	1	3	40	21	33	184 + 5:	189.

Different cause: 12 × "last eye".
Different site: 18 × "last eye", 5 × right eye.

Table A 28. 204 blind men, over 40 years of age, seen at Undaan 1961. Visual acuity less than $1^1/_2/60$. Causes of blindness by site/type and etiology.

	12	12 /19	19 .9	20	20 .1	28 04	40 08	46	62	91	98	99	Total	
110			1	2					1	38			42	
130						1							1	
212	2												2	
313				34	3	2	1				1	12	53	
315		1	2	2	1					:1	6	16	29	: Pemphigus
316											1		1	
320								1				1	2	+ 19.9/313-510
370.1											1		1	19.9/315-510
380.4				1								2	3	19.9/315-710
380.5												1	1	62/410-110
410			1	2						22			25	91/410-420
480										1			1	98/710-315
510			3	1					2		7		13	99/315-530
550											1		1	91/110-99/315
560			6								1		7	99/315-91/110
610			1										1	99/315-91/410.
660											1		1	
710			8								2		10	
Total	2	1	22	42	4	3	1	1	3	62	21	32	194 + 10:	204.

Different cause: 7 × "last eye".
Different site: 20 × "last eye", 8 × right eye.

Table A 29. 212 blind men, over 40 years of age, seen at Undaan 1962. Visual acuity less than $1^1/_2/60$. Causes of blindness by site/type and etiology.

	12/98	19.9	20	20.1	21/91	28 03	28 04	62	62/19	82	91	98	99	Total	+
110			2								27			29	91/410-420
150	1											2	2	5	91/420-410
212	2													2	46.09/150-313
313		1	23				1					3	11	39	
315		3	3	1								6	26	39	91/410-49.08/313.
380.4		1	3			1							3	8	
380.5												1		1	
410		5	2		1			3			28			39	
420					1									1	
510		4										6		10	
550												1		1	
560		5										1		6	
610		1	1							2				4	
650										4				4	
710		13							1			3		17	
810												1		1	
990													2	2	
Total	3	33	34	1	2	1	1	3	1	6	55	24	44	208 + 4 :	212.

Different cause : 12 × "last eye".
Different site : 15 × "last eye", 34 × right eye.

Table A 30. 120 blind men, over 40 years of age, seen at Undaan 1/8'66-1/8'67. Visual acuity less than $1^1/_2/60$. Causes of blindness by site/type and etiology.

	12	12?	12/19	19	20	40	46	62	62/98	89/98	91	98	99	Total	+
110											22			22	91/410-98/315
122											1			1	91/410-99/315 (2×)
130						1							2	3	98/313-91/410
212	1													1	
311												1	1	2	
313				2	7								6	15	
315		1			4		1						7	13	
316	1		1											2	
380.4					1								4	5	
380.5		1											2	3	
410					2		1	2	1		15			21	
420													1	1	
510				3								1		4	
530													1	1	
550				1								2		3	
560												1	1	2	
640												3		3	
650										3				3	
660												4		4	
710				6										6	
810												1		1	
Total	2	2	1	12	14	1	2	2	1	3	38	13	25	116 + 4 :	120.

(2 blind men already in the 1958-62 lists came again during this year).

Table B 5. 942 blind men, over 40 years of age, seen at Undaan 1958-62. Visual acuity less than $1^{1}/_{2}/60$. Causes of blindness by site/type and etiology. Compilation of Tables A 25-29.

	12	18	19	20	21	27	28	40	46	49	62	65	81	91	98	99	Total
110			2	5							1			146			154
122															1		1
130				1			1									2	4
150	1									1					2	14	18
190																1	1
212	15																15
311			1	2											2	1	6
313	1		1	165		1	4	1		1					7	44	225
315	2	2	8	12								1		1	18	107	151
316			2	1											1	4	8
319			1														1
320									1							1	2
370			1	3											2		6
380			1	10			1								2	21	35
410			11	11	1						4			60	3		90
420			1		1										1		3
480														1			1
510			22	2							2				26	3	55
530																1	1
550			7												4		11
560			20									1			4		25
580															1		1
590															1		1
610			4	1							1		2		4		12
640															2		2
650													12				12
660															1	1	2
710			51	1							1				11	1	65
810															1		1
990																3	3
Total	19	2	133	214	2	1	6	1	1	2	9	2	14	208	94	204	912 + 30 : 942

If the right eye's classification of 30 persons not included in the above tabulation (see Tables **A** 25-29) is taken as the decisive one, the following **summary** of causes of blindness in men over 40 years of age can be given:

Site. Eyeball 178 + 6:	184	19.5%
Conjunctiva 15	15	1.5%
Cornea 434 + 10:	444	47%
Lens 94 + 7:	101	10.7%
Uvea 94 + 6:	100	10.7%
Retina 28	28	2.9%
Optic nerve 65 + 1:	66	7%
Other 4	4	0.4%

Etiology. Gonorrhoea 19	19	2%
Syphilis 133 + 7:	140	14.8%
Trachoma 214	214	22.7%
Trauma 4 + 1:	5	0.5%
Congenital 14	14	1.5%
Glaucoma 147 + 5:	152	16%
Cataract 67 + 7:	74	7.8%
Ignota, Wr — 91 + 4:	95	10.6%
Ignota 204 + 6:	210	22.6%

Summary Of Table A 30, 120 blind men over 40 years of age, 1/8'66-1/8'67. Visual acuity less than $1^1/_2/60$.

Site. Eyeball	26	21.7%
Conjunctiva	1	0.8%
Cornea	41	34.1%
Lens	25	20.8%
Uvea	10	8.3%
Retina	10	8.3%
Optic nerve	6	5%
Other	1	0.8%

Etiology. Gonorrhoea	5	4.1%
Syphilis	12	10%
Trachoma	14	11.6%
Trauma	3	2.5%
Congenital	3	2.5%
Glaucoma	22	18.3%
Senile cataract	25	20.8%
Ignota, Wr —	14	11.6%
Ignota	25	20.8%

Table A 31. 160 women. over 40 years of age, seen at Undaan 1958. Visual acuity less than $1^1/_2/60$. Causes of blindness by site/type and etiology.

	12	19 .1	19 .9	20	20 .1	28 04	49	63	67	91	98	99	Total	
														+
110										41			41	20.1/510-311
150				1								1	2	99/380.4-510
212	7												7	99/510-315
280				1									1	99/530-380.5
311												2	2	
313			1	37		1			1		1	9	50	20/313-99/510
315				1							1	16	18	98/510-91/110
316												3	3	99/315-91/110(2×)
320			1										1	
370.1				2									2	
380.2					1								1	
380.3											1		1	
380.4				1							1	1	3	
380.5												2	2	
380							1						1	
410		1	1	2				1		1			6	
510											1	2	3	
530					1						1		2	
550			2										2	
710			3										3	
810			1										1	
Total	7	1	9	45	2	1	1	1	1	42	6	36	152 + 8 : 160.	

Different cause : 6×"last eye".
Different site : 10×"last eye", 9× right eye.

Table A 32. 198 women, over 40 years of age, seen at Undaan 1959. Visual acuity less than $1^1/_2/60$. Causes of blindness by site/type and etiology.

	12	12 /19	19 .9	20	20 .1	64	91	98	99	Total	
											+
110							65			65	12/212-370
122			1							1	20.1/315-510
130				1					1	2	98/315-510(2×)
150	1								1	2	98/510-380.5
212	8	1								9	99/315-510
311			1							1	
313	1		2	39	5			1	6	54	20/313-91/420
315			1					2	15	18	91/110-99/315(4×).
316								1	1	2	
370.1				2	1					3	
380.4			1	1					2	4	
380.5									1	1	
410			2		1		1	1		5	
420							1		1	2	
510			3					3	2	8	
550			2					1		3	
710			5			1				6	
810			1							1	
Total	10	1	19	43	7	1	67	9	30	187 + 11: 198.	

Different cause : 16×"last eye".
Different site : 16×"last eye", 11× right eye.

Table A 33. 214 blind women, over 40 years of age, seen at Undaan 1960. Visual acuity less than $1^1/_2/60$. Causes of blindness by site/type and etiology.

	12	19 .9	20	20 .1	28 01	62	65 .8	82	91	98	99	Total	+
110			1						59			60	98/315-530
122									1			1	98/510-313
130										2		2	98/510-315
150											8	8	99/710-315
212	8											8	
311			1	1								2	91/110-99/313
313		1	39	5						4	7	56	20.1/316-91/410
315			3	1	1						12	17	99/315-91/110(2×).
316		1										1	
370.1			3								1	4	
380.4			2									2	
380.5										2		2	
390									1			1	
410		1	2		1	1			13			18	
510										7	5	12	
530		1								1		2	
560		1										1	
640										1		1	
650								1				1	
710		6					1					7	
Total	8	11	51	7	2	1	1	1	74	17	33	206 + 8 :	214.

Different cause : 12×"last eye".
Different site : 18×"last eye", 6× right eye.

Table A 34. 208 blind women, over 40 years of age, seen at Undaan 1961. Visual acuity less than $1^1/_2/60$. Causes of blindness by site/type and etiology.

	12	12 /19	19 .9	20	20 .1	28 04	40 /19	82	91	98	99	Total	+
110				1					68			69	20/370.1-410
130				1							1	2	98/315-510(3×)
150											1	1	
212	4	1										5	91/110-99/315(2×)
313	1		1	41	1	2				3	3	52	91/410-99/315
315	1			2						2	16	21	99/315-91/110.
370.1				2								2	
380.4				3	1						2	6	
380.5				1						1	1	3	
410				3					14			17	
420									3			3	
510										1	2	3	
530			1							2		3	
550			1							2		3	
560			2									2	
570							1					1	
610			1							1		2	
650								2				2	
710			2							1		3	
Total	6	1	8	59	2	2	1	2	85	13	26	200 + 8 :	208.

Different cause : 14×"last eye".
Different site : 13×"last eye", 8× right eye.

Table A 35. 211 women, over 40 years of age, seen at Undaan 1962. Visual acuity less than $1^1/_2/60$. Causes of blindness by site/type and etiology.

	12	18 /20	19 .9	20	20 .1	82	91	98	99	Total	
											+
110							54			54	19.9/313-510
130	1									1	20.1/370.1-530
150									3	3	98/315-370.1
212	2									2	98/410-660
313	2	1		37	2			3	3	48	98/510-313
315	1							4	12	17	99/315-110
316				1						1	
370.1				2						2	20/313-99/530
380.4				3					1	4	20/313-91/110
380.5				1				1		2	46/130-99/315
410			1	8			26			35	49/180-91/410
420							2			2	49/510-91/410
510								4		4	91/110-12/212
530								1		1	98/530-91/410
550								1		1	99/315-91/110(2×)
650						4				4	99/380.5-91/110.
710			8					6	1	15	
Total	6	1	9	52	2	4	75	20	20	195 + 16 : 211.	

Different cause : 17×"last eye".
Different site : 21×"last eye", 10 × right eye.

Table A 36. 111 blind women, over 40 years of age, seen at Undaan 1/8'66-1/8'67. Visual acuity less than $1^1/_2/60$. Causes of blindness by site/type and etiology.

	12	19	20	28	40	62	91	98	99	Total
110			5				44			49
122							2			2
130					1					1
150	1								1	2
212	1									1
313			13	1				1	1	16
315	3								5	8
316	1									1
370			2							2
380.4			1					1		2
390									1	1
410			5			1	11			17
510		1								1
560		1								1
570							1			1
710		3						3		6
Total	6	5	26	1	1	1	58	5	8	111

(4 blind persons already in the 1958-62 lists came again during this year).

Table B 6. 991 blind women, over 40 years of age, seen at Undaan 1958 - 62. Visual acuity less than $1^1/_2/60$. Causes of blindness by site/type and etiology. Compilation of Tables A 31 - 35.

	12	18	19	20	28	40	49	62	63	64	65	67	82	91	98	99	Total
110				2										287			289
122			1											1			2
130	1			2											2	2	7
150	1			1												14	16
212	31																31
280				1													1
311			1	2												2	5
313	4	1	5	206	3							1			12	28	260
315	2		1	7	1										9	71	91
316			1	1											1	4	7
320			1														1
370				12												1	13
380			1	14			1								6	10	32
390														1			1
410			6	16	1			1	1					55			80
420														6		1	7
510			3												16	11	30
530			2	1											5		8
550			5												4		9
560			3														3
570						1											1
610			1												1		2
640															1		1
650													7				7
710			24							1	1				7	1	34
810			2														2
Total	39	1	57	265	5	1	1	1	1	1	1	1	7	350	64	145	940 + 51 : 991

If the classification for the right eye of 51 persons not included in the above cross-tabulation (Tables A 31 - 35) is taken as the decisive one, the following **summary** can be given for women over 40 years of age:

Site. Eyeball	314 + 10:	324	32.6%
Conjunctiva	32 + 1:	33	3.3%
Cornea	410 + 27:	437	44 %
Lens	78 + 2:	89	8.9%
Uvea	51 + 10:	61	6.1%
Retina	10	10	1%
Optic nerve	34 + 1:	35	3.5%
Other	2	2	0,2%

Etiology. Gonorrhoea	39 + 1:	40	4 %
Syphilis	57 + 1:	58	5.8%
Trachoma	265 + 9:	274	27 %
Trauma	2 + 3:	5	0.5%
Congenital	7	7	0.7%
Glaucoma	287 + 8:	295	29.7%
Senile cataract	80 + 2:	82	8.2%
Ignota, Wr —	63 + 14:	77	7.7%
Ignota	145 + 14:	159	16 %

Summary of Table A 36, 111 blind women over 40 years of age, seen 1/8'66 - 1/8'67.

Site. Eyeball	54	48.6%
Conjunctiva	1	0.9%
Cornea	30	27 %
Lens	17	15.3%
Uvea	3	2.7%
Retina	—	—
Optic nerve	6	5.4%
Other	—	—

Etiology. Gonorrhoea	6	5.4%
Syphilis	5	4.5%
Trachoma	26	23.4%
Trauma	1	0.9%
Congenital	—	—
Glaucoma	44	39.6%
Senile cataract	12	10.8%
Ignota, Wr —	5	4.6%
Ignota	8	7.2%

Chapter 13. Discussion of Causes of blindness and their relative Importance.

In the preceding chapters first the data on causes of blindness are given according to sex, age and year of first examination, arranged in cross-tabulations, using the International Standard Classification (Tables marked A). Each of these groups has been compiled again in Tables marked B, giving only the cases, where essentially the same cause was responsible for blindness in the two eyes. In Table B 7 are added the data from the A-lists which concern cases where blindness in the two eyes was caused by a distinctly different cause. The special causes which have some importance only in this group are : Variola and Trauma.

From the data collected there were calculated percentages and they were arranged in the same way as is done by A. Sorsby in his ”The Incidence and Causes of Blindness in England and Wales 1948—1962” using especially those tables which concern the same period of time as my investigation. These tables were marked ”C”.

There are 3 main groups of C-tables : C1, C2, C3, C4 bring the data for the 1958—1962 period, limit of visual acuity $1^1/_2$60.

C5, C7, C8, C10 : data for 1/8’66—1/8’67, limit of visual acuity $1^1/_2$/60.

C6, C9, C11, C12 : data for same period, limit of visual acuity 1/10.

Two additional groups of C-tables concern data for 1/8’66—1/8’67 where cataract cases which did not yet undergo surgery are included (see later on) :

C13, C15, C16, C19 : limit of visual acuity $1^1/_2$/60.

C14, C17, C18, C20 : limit of visual acuity 1/10.

Finally C21, C22, C23, C24 concern monocular blindness in the period 1/8’66—1/8’67, again using the 2 different limits of visual acuity.

For this discussion the same type of C—tables is compared for 1958—62, for both limits of visual acuity in the period 1/8’67—1/8’67, and with both limits of visual acuity in the period 1/8’67—1/8’/67, and with the same type of Tables in Sorsby’s book.

Tables C1, C5 and C6 (Table 7 in Sorsby) give the percentages first of the causes of blindness by **site/type** of eye affection, arranged as in the International Standard Classification. In all age groups, for both sexes, and as well in the earlier as in the later period of investigation, the **Corneal** site always has the highest percentage. In children this percentage is even higher than in adults. Next in frequency comes ”**Eyeball as a whole**” (which included i.a. Glaucoma) in all age groups. In the middle age groups the **uveal** and the **optic nerve** site are next in importance whereas in the oldest age group the **lens** becomes the most important in frequency.

In the same Tables are mentioned the **etiologic** causes of blindness, again arranged according to the International Standard Classification, which gives only a rough division. In children ”**systemic diseases**” (mostly Keratomalacia) is consistently in all periods the most frequent cause of blindness. In the middle age groups the most frequent etiological cause is ”**undertermined**”, which includes Cataract and Glaucoma, followed by ”infections”, which include Trachoma, Syphilis and Gonorrhoea. The same goes for the oldest age group. It should again be stressed, that ”undetermined” does not mean ”unknown cause”, but only etiologically undetermined : cataract and glaucoma, the most frequent eye diseases entering in this group, have as yet no known etiological cause, but as eye diseases they are quite circumscript.

If we compare these data with those given in Sorsby's Table 7, the difference between a developed country like England and Wales, where the **retina** and the **lens** are the most frequent sites of causes of blindness, and where **"undetermined"** and **"systemic diseases"** are the most frequent etiological factors, and an underdeveloped region like the one in the present study is very clear.

Tables C2, C7, C9 (Sorsby's Table 8) give lists of important clinical entities responsible for blindness, by sex. I have been obliged to add 3 items to Sorsby's list, viz. **Trachoma, Keratomalacia** and **conjunctivitis gonorrhoica nonneonatorum,** because each of these entities causes more than 5% of the blindness in both sexes. From this table it appears that there is a female preponderance for Trachoma, even more for Glaucoma, a male preponderance for Keratomalacia, and less for Choroiditis and Optic Atrophy. If we compare the earlier period with the later one, there seems to be an increase in : Glaucoma, Cataract, and Keratomalacia, and on the other hand a decrease in : uveitis, Trachoma and optic atrophy. Comparing the group with visual acuity limit of $1^1/_2/60$ with that of 1/10 during the one year-period 66/67, we discover higher percentages of very low visual acuity in : glaucoma, optic atrophy, Keratomalacia and conjunctivitis gonorrhoica. Cataract and Trachoma on the other hand often do not damage visual acuity to a maximal extent.

On comparing these figures with those given by Sorsby in his Table 8 (not copied here, as essential data can be found in his Table 9, see later on) we find that quite a few clinical entities which are important enough in England are unimportant here or even non-existent, e.g. toxoplasmosis, retrolental fibroplasia, diabetic retinopathy, and the group hypertensive and vascular retinopathy. Diabetic and vascular retinopathy do occur here, but they only gain importance in countries where the life expectancy is high, and maybe also where the standard of living is high. The poor population in and around Surabaja does not live long enough and probably has not enough to eat to develop diabetes and vascular disease lasting so long that it causes blindness by retinopathy.

Tables C3, C8, C11 (Sorsby's Table9) show various causes of blindness in age groups by sex. Naturally the importance of congenital causes of blindness decreases with age. Keratomalacia is only mentioned in the youngest age group : for the older age group it is included in disease of the whole eyeball or of the cornea by unknown cause, as it is often impossible after many years have passed, to ascertain the differential etiological diagnosis between Keratomalacia, Conjunctivitis Gonorrhoica and other causes in the many cases of : Phthisis bulbi, Staphyloma corneae and Leucoma corneae.

Trachoma, Cataract and Glaucoma increase in importance with age. In a diagram (Fig. G) the percentages of the causes of blindness as stated in Table C3 and C8 are illustrated. The 5 year-period 1958/62 is compared to the one year-period 66/67. From this diagram it appears that Keratomalacia and Glaucoma are increasing in recent years, whereas corneal disease (excluding Trachoma, Keratomalacia, Conj. gonorrh. and genetic) are decreasing.

Compared to Sorsby's figures in his Table 9, there appears a distinct difference in the youngest age group, which in England shows a percentage of 56% for congenital malformations, here only of 15%. This difference is probably caused by the lack of other important causes of blind-

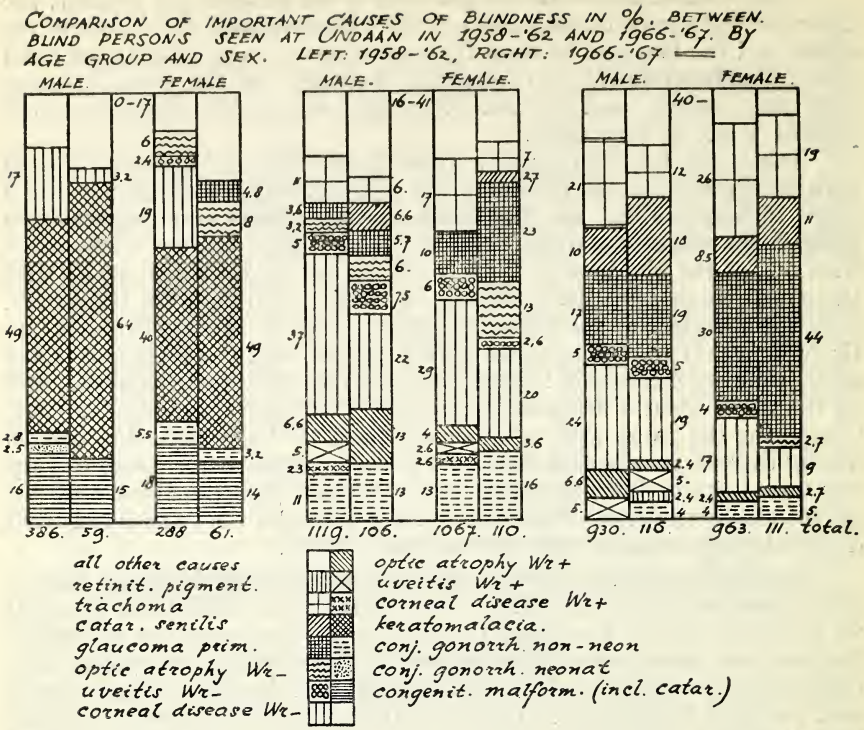


Fig. G

ness at an early age in England; especially Keratomalacia and conjunctivitis gonorrhoica cause so much blindness in and around Surabaja, that congenital malformations which also occur, necessarily show a lower relative frequency. It is to be expected that if and when conditions here are improving so that these two diseases disappear from the scene, congenital malformations will take first place like in England at the present. In the case of other differences, which are less striking, the same argument probably can be applied.

Tables C4, C9, C20 : using the same figures for the composition of the population in general which have been used in chapter six, the percentages of the most frequent causes of blindness were adjusted, stating in the Tables the adjusted female incidence and the actual male incidence. In this way the overall surplus of women in the general population could be compensated for. The rate of adjustment is mentioned in the tables, and differs for the various age groups, and is also not the same for 1961 and 1966. By these adjustments it becomes clear that some apparent differences in incidence between male and female are not real. If tested by the X^2 method, it appears that only the following differences are statistically significant :

1. Keratomalacia: boys are more often blinded by this cause than girls (significant at 5% level).
2. Glaucoma in females over 16 yrs. of age is more often the cause of blindness than in males (0,5% level).

3. Trachoma in females over 16 yrs. of age is more often causing blindness than in males (0.5% level).

4. Syphilitie optic atrophy in males over 16 yrs. of age is a more frequent cause of blindness than in iemales (0.5% level).

Significance has been calculated for the apparent male preponderance in: congenital malformations, retinoblastoma, and retinitis pigmentosa: the differences were non-significant. For cataract also there was no statistically significant difference between the sexes.

These calculations have been performed for mean values of the period 1958/62.

Comparing them with the figures from Sorsby's Table 10, it appears that in England the female preponderance for glaucoma disappears on adjustment, but in the region of Surabaja the female preponderance for glaucoma is so strong, that it survives as well adjustment according to the general population as the calculations for statistical significance.

In the next Table 10 a few of the important blinding diseases are compiled as frequencies for the 1958/62 period and the 66/67 period, using the year '58 and the year 66/67 for statistical comparison.

Table 10 Trends in Frequency of causes of blindness, Undaan 1958/62 and 1966/67.

Males 0-17 yrs	'58 91	'59 100	'60 77	'61 74	'62 60	'66/67 59 total
Gonorrh.	2 2.2%	9 9%	3 3.9%	4 5.4%	3 5%	1 1.7 %
Keratomal.	43 47 %	53 53%	41 53 %	31 42 %	17 27%	38 64.4%
Ignota	26 28.5%	19 19%	15 19 %	14 18 %	13 22%	3 5.1 %
Females, 0-17 yr	69	66	49	56	58	61 total
Gonorrh.	8 11%	7 10.7%	4 8%	2 3.5%	1 1.7%	2 3.2 %
Keratomal.	27 39%	34 51.5%	15 31%	23 41 %	16 28 %	30 49 %
Ignota	19 24%	12 18 %	10 20%	14 25 %	9 16 %	7 11.3%
Males 16-41 yr	284	246	214	205	190	106 total
Gonorrh.	30 10.5%	40 16.2%	22 10 %	26 12.6%	25 13 %	17 16 %
Syphilis	36 12.7%	36 14 %	30 14 %	30 18.5%	14 7.3%	17 16 %
Trachoma	31 11 %	24 9.7%	23 10.7%	30 18.5%	14 7.3%	5 4.7%
Glaucoma	12 4.2%	10 4 %	9 4.2%	14 6.8%	8 4.2%	6 5.6%
Ignota	159 55.3%	117 43.5%	98 46 %	85 41.4%	82 43 %	38 33.4%
Females 16-41 yr	232	231	223	218	187	111 total
Gonorrh.	25 10.7%	39 16.7%	31 14 %	30 13.7%	22 11.8%	18 16 %
Syphilis	17 7.3%	21 9 %	22 10 %	25 11.4%	19 10 %	8 7.2%
Trachoma	34 14.6%	38 16 %	42 18.8%	40 18.3%	28 15 %	8 7.2 %
Glaucoma	19 8.1%	17 7.3%	25 11.2%	18 8.2%	23 12 %	25 22.5%
Ignota	107 46 %	94 40.7%	84 37.6%	74 40 %	69 37 %	44 39.6%

Males over 40 yr	178	159	189	204	212	120 total
Gonorrh.	5 2.8%	5 3.1%	3 1.6%	3 1.4%	3 1.4%	3 2.5%
Syphilis	26 14.6%	23 15 %	27 14.3%	22 20.7%	33 16 %	12 10 %
Trachoma	44 25.3%	38 24 %	51 27.4%	46 22.5%	35 16.5%	14 11.6%
Glaucoma	24 13.5%	21 13.2%	36 19 %	39 19 %	27 12.7%	22 18.3%
Ignota	69 38.7%	54 34 %	54 28.5%	53 26 %	68 32.7%	38 31.6%
Females over 40 yr	160	198	214	208	211	111 total
Gonorrh.	7 4.3%	1 5.5%	8 3.6%	7 7.3%	6 2.8%	6 5.4%
Syphilis	10 6.2%	19 9.5%	11 5 %	8 3.8%	9 4.2%	5 4.5%
Trachoma	47 29.3%	50 25.2%	58 27 %	61 29.3%	54 25.5%	26 23.4%
Glaucoma	41 25.6%	65 32.8%	59 27.5%	68 32.7%	54 25.5%	44 39.6%
Ignota	42 26.2%	39 19.2%	50 23.3%	39 18.2%	40 19 %	13 11.7%

From this table 10 the statistical significance of some trends was calculated by the X^2 method.

1. Increase of Keratomalacia between 1958 and 66/67 was significant for 0.5% level in boys, not significant in girls.

2. For men between 16 and 41 yrs. of age the decrease of Trachoma and the increase of Glaucoma was not statistically significant.

3. For women between 16 and 41 yrs. of age the decrease of Trachoma was not significant, the increase of Glaucoma was significant for 0.5% level.

4. For men over 40 yrs. of age the decrease of Trachoma was significant for 2.5% level. The increase of glaucoma was not significant.

5. For women over 40 yrs. of age the decrease of Trachoma was significant at 5% level. The increase of Glaucoma was significant at 2.5% level.

6. The figures for conjunctivitis gonorrhoica were apparently slightly decreasing, but they showed many ups and downs in the interlying years, so it was considered not to be justified to assume a real "trend" in any age group.

7. The same applied to Syphilis in the age groups above 16 yrs. There seemed to be some decrease, but again the intervening years showed so many ups and downs, thet a "trend" could not be ascertained from these figures.

Trends in frequency of eye diseases as seen in the outpatients department.

To ascertain if these trends only were valid for eye diseases as causes of blindness, or if they were more generally effective, I collected the figures for some of the more important eye diseases as they were found in the outpatients' department of Undaan Eye Hospital, including all patients, not only those who became blind.

Table 11 Frequency of some eye diseases in 1956 and in 1966, Undaan.

	1956		1966	
	11410 adults	2405 children	11066 adults	3232 children
Trachoma	23 %	14 %	11 %	6 %
Conj. gonorrh.	1.1%	2.5%	0.65%	1.4%
Xerophthalmia		11,7%		18.4%
Glaucoma	2.2%		2. 1%	
Cataract	4.7%		6.3%	
Children and adults together :				
Conj. gonorrh.	1.4%		0.76%	
Xerophthalmia	2 %		3.8 %	

If tested with the X^2 method, the statistical significance of the various trends is as follows :

1. The decrease of cenjunctivitis gonorrhoica is significant for the 0.5% level as well for children as for adults, and also for both taken together.

2. The decrease of Trachoma is significant for 0.5% level, as well in children as in adults.

3. The increase in Xerophthalmia (not only Keratomalacia) in children is significant at 0.5% level, even if children and adults are taken together.

4. The increase of cataract in adults is significant at 0.5% level.

Senile cataract. The increase in the figure for the percentage of cataract, is partly to be ascribed to the decrease of other causes of blindness. If compared to the figures as listed by Sorsby, the main difference is, that in England cataract as a blinding disease gains importance at a later age : our figures have already some importance before the age of 40, in Sorsby's list cataract emerges only after age 50. This is in accordance with the literature where generally is stated that cataract in hot countries strikes at an earlier age than in temperate zones. This is possibly caused by earlier aging, and possibly by other as yet unknown factors.

The percentage in the period 1958/62 contain only cases where either surgery was refused, where there was a contraindication, or where surgery was unsuccessful. As our cases were seen in an ophthalmological hospital it was natural that patients as well as doctors were willing to proceed to surgery in as many cases as possible. The British figures on the other hand are based on data not only from ophthalmologists, but also from general practitioners, and for the earlier periods even from laymen. Even in England there seems still to exist a reluctance against cataract surgery in old people, all of which factors cause an unexpectedly high frequency of senile cataract in Sorsby's lists, which is much higher than e.g. in Holland (Schappert-Kimmeyser, p.53.414) where cataract as a cause of blindness is found in 5%.

Life expectancy which is of course an important factor in the chance for cataract to develop, is much lower in Indonesia, only partly compensated for by the younger age at which cataract is already found.

In an effort to collect figures on the frequency of cataract which were more comparable to the British ones, I noted for the year '66/'67 also the cases of cataract, which were not (yet?) operated upon, and in that percentages were nearer to those found in England. Anyway, it is certain that cataract here is a common cause of blindness in older people, but it is not as frequent as is sometimes described from other areas in hot climates.

Monocular blindness.

Cases of blindness in one eye only, where the other eye was still good, I collected only for the one year-period 1/8'66-1/8'67, Tables A43 through 54, one list using the limit of visual acuity $1^1/_2/60$, and the other 1/10. Tables C21 through 24 were based on these data.

As was seen in blindness with a different cause in the two eyes, in the case of monocular blindness also some special causes gain importance, viz.

a. Trauma, which is more frequent as a cause of monocular blindness than of binocular blindness, and more frequent in males than in females, causing 27% of monocular blindness in boys under 17, 20% in younger men, and 7.6% in older men.

b. Retinoblastoma also causes more often monocular blindness than binocular: 6% of monocular blindness in boys, and 8% in girls is caused by it.

Other important blinding diseases cause less often monocular blindness than binocular blindness.

c. Keratomalacia causes 31% in boys and 40% in girls.

d. Glaucoma causes 10% in older men, 18% in older women.

e. Cataract causes 7% in men and women between 16 and 41 yrs. of age, and 40% in men and women of over 40 yrs. of age.

f. Optic atrophy (all etiological causes) 0.4% in men, 1.5% in women of 16-41 yrs. 4.4% in men, 1% in women over 40 yrs.

g. Trachoma : 2% in boys, 1.3% in girls under 17 yrs. of age, 3.7% in men, 9% in women between 16 and 41 yrs. of age, 2.6% in men, 4.5% in women over 40 yrs. of age.

This is as could be expected. Only the most illustrative types of C — Tables have been calculated for monocular blindness.

Conclusions :

From the figures, as well those relating to blinding eye diseases, as to frequencies as seen in the outpatients' department, it can be concluded that generally speaking :

1. Trachoma is losing in importance in recent years, although no special effort was devoted to control by mass campaigns or by school treatment in the period covered in this study. Probably a general rise in hygiene has its effect, which given the character of the disease cannot be swift.

2. Xerophthalmia is becoming more frequent generally, and its blinding form, Keratomalacia, especially in boys, is on the increase. Various socio-economic factors can be cited as the cause of this unfavorable development in spite of the eradication of malaria, combined with a persistent ignorance relating to appropriate feeding of infants and children.

3. Glaucoma is on the increase, especially among women. Partly this is caused by a higher life expectancy by the eradication of malaria and better treatment of other infectious diseases, and partly the increase is probably apparent only, because even older women now are less reluctant to consult a doctor than they have been in former times.

4. Syphilis as a cause of blindness does not yet show a clear tendency to decrease. In the eradication mass campaigns against Yaws which were carried out in this region in ca. 1954-1958 the penicillin treatment must have prevented development of a number of syphilitic infections also. As the period between infection and the development of tabetic optic atrophy is ca. 12-16 years, it can be hoped, that in the next years a decrease in this cause of blindness will develop as an unintended result of the mass campaigns against Yaws.

Chapter 14. Previous investigations into the Causes of Blindness in Indonesia.

One of the very early pioneers in the field of ophthalmology in Indonesia has been Leo Steiner, who after service in the Navy in 1892 took up the civil post of tweede stadsgeneesheer (second town doctor) at Surabaja. He had trained as an oculist in Switzerland and Austria, and stayed at Surabaja until 1914. He began to help poor patients without remuneration, a job which was carried on by his successor Dr. A. Deutman, who founded in 1916 with other health authorities and businessmen the Association which until now runs the Undaan Eye Hospital. After Deutman was interned by the Japanese in 1942 Dr. I.H. Go carried on, eventually seconded by me ; after the departure of Go in 1954 I succeeded him, and was in 1955 seconded by dr. Moh. Basoeki, who changed places with me in 1959, taking over the leadership. In this short summary of the history of Undaan Eye Hospital one fact stands out, i.e. the continuity from Steiner's early beginnings until the present time.

In 1894 Steiner, impressed by the number of blind people he saw among his patients, surveyed 95, 187 persons in the poorer parts of the town (Kawedanan Kapasan, Krembangan, Kranggan, Njamplungan and part of Kupang). Among them he found 504 blind persons, only 16 children among them. He supposes that many children who had been blinded by conjunctivitis gonorrhoica soon may have died from sheer misery. To the blind persons discovered in his survey he added 156 encountered in the dispensary, and found as the cause for their blindness :

Trachoma	37.73%	Glaucoma	5 %
Blennorrhoea	18.65%	Cataract	1.97%
Variola	1.21%	Complicated Cataract	3.48%
Leucoma corneae & Phthisis bulbi from other causes	22.27%	Optic nerve atrophy	2.88%
		Retinal detachment	0.45%
		Amaurosis	1.97%
Keratit, parench.	0.45%		
Sympath. ophth.	0.61%		
Iritis, iridocycl.	3.03%		

His comments: Trachoma is a disease of the poor, whereas Blennorhoea is seen more evenly spread over all social strata. Variola caused very little blindness, as a consequence of the efficient system of vaccination. He estimates that at Surabaja there were 670 blind persons among a Native population of 128.294. From this incidence he generalizes that for the whole of the islands Java and Madura (23.956.962 inhabitants) there must be 125.000 blind persons. From the list of causes he concludes that 80% of the blindness would be preventable, and enters a plea for more interest in this problem. Fully 70 years later we can still repeat the same plea.

Another point which Steiner makes and which is still valid, is that it is often very difficult to ascertain the cause of blindness in inveterate cases. In his list he brought casec who were blinded by different causes in both eyes under the cause of the eye which was blinded last. His definition of blindness: inability to find one's way about without help, ca. counting fingers at 0.75 metre.

An interesting point is, if we look at his figures, that Keratomalacia is not present. From a report in 1896 there emerge: 1 case of Xerosis conjunctivae and 3 cases of Hemeralopia. Literally hundreds of cases of Leucoma corneae, phthisis bulbi where the cause cannot be ascertained (479) and which possibly are to be ascribed to Keratomalacia at least in part.

From various sources we can read that in the first decade of the century there was a relatively high number of eye patients who sought help. From the Mission hospital in the village Modjowarno, which was fouded in 1894, the annual reports for 1906, 1908, 1909, 1911 state high percentages of blind persons, in 1906 even 9% of all cases, in 1908 264 blind among 1206 eye patients. From other hospitals also we get the impression that the majority of patients were suffering from eye or skin diseases (100.). At Ngawi, at the border between East and Mid Java, in 1903 there was opened a dispensary with the special goal of doing something for the eye patients in that region: in 5 years' time there were seen over 5000 eye patients (1909. 590).

At Bandung, West Java, in 1901 an Institute for the Blind was opened, stimulated by the activity of the oculist Dr. Westhoff. According to him (563) 50% of the inmates were blinded by conjunctivitis gonorrhoica.

Louwerier (1923. 245) reported on his experience during the second decade of this century in an outpatients' dept. on Sumatra's East Coast, at the time a region where medical progress was quick and effective. From his list of causes of blindness I give only the figures relating to Malayans (natives of Sumatra), Javanese (imported labor) and Chinese, for which as well cases of monocular as of binocular blindness are given:

	Malay		**Javanese**		**Chinese**	
	1 eye	2 eyes	1 eye	2 eyes	1 eye	2 eyes
Leucoma corneae	30	53	7	16	44	32
Staphyloma corneae	10	12	2	2	16	10
Iritis	8	9	3	7	10	5
Choroiditis	2	2	1	1	—	1
Optic atrophy	1	21	1	12	7	32
Retinal detachment	—	—	1	—	3	1
Glaucoma	4	6	3	3	6	14
Amblyopia ex anopsia	1	1	2	1	6	1

Retinitis pigmentosa	—	—	—	—	—	—
Trauma	2	—	1	—	2	—
Phthisis bulbi	52	60	15	10	50	40
Anophthalmus	9	—	6	—	17	—

Etiological factors are not stated.

The census of the whole population which was carried out in 1920 included a count of the blind. Bakker gives the following figures (18) :

Total number of blind persons in the country : 75.000.

Incidence rate on **Java**		on the **other islands**
Natives	1.61 promille	1.26 pro mille
Europeans	0.83 ,,	0.3 ,, ,,
Other Orientals	0.865 ,,	0.45 ,, ,,

According to Mulock Houwer (311) the 1920 census leaves room for doubt, the 1930 census was more trustworthy, as is also stated by Tyssen (1940/951) because they had a clear instruction as to the definition of blindness (inability to see fingers in front of the eyes). The 1930 census showed ca. 132.000 blind persons, more than 100.000 on Java and Madura alone.

According to de Ruyter (1923. 405) there were in the Residency of Cheribon in 1920 211 blind persons among 62.000 inhabitants (201 verified by a physician) i.e. 35 promille.

In the thirties of this century the "travelling eye doctor" Tyssen worked on Sumatra, Java and Celebes, and gave broad publicity to his findings (520/524). But precisely because he travelled, many of his impressions were not founded on exact data. By others in certain regions surveys were made which tried to collect the real figures of eye diseases and blindness. Gardjito (137) published figures on his experience in the region of Pemalang, where a trachoma campaign was organized : between 1930 and 1937 among 1750 families in 3 villages there were found 37 blind persons, 25 of whom were blind by Trachoma.

Stimulated by the publicity given to the work of Tyssen the Dutch organization Simavi paid the salary for an oculist who worked intensively in part of Sumatra, and who collected data on 3079 eye patients, of whom 381 were blind and 535 blind in one eye (van Manen 1938. 262). As he uses a somewhat complicated system of classifying patients who have gone blind by a different cause in each eye, and because it is not exactly stated in which type of cases the patient could be helped by surgery, it is not possible to calculate percentages. From the figures it is anyway clear, that for young children and adolescents Xerophthalmia is an important blinding disease, for middle age groups Trachoma and Cataract as well as Optic nerve atrophy, and some Glaucoma. This continues into old age (over 50). Conjunctivitis gonorrhoica is wery rare or nonexistent on Sumatra. I give van Manen's table, leaving out the monocular cases.

Table 7 Causes of blindness, by age and sex. J.G. van Manen, 1937, Tapanuli district (Sumatra) and Nias Island.

Cause	0-1¾ yr M	0-1¾ yr F	1¾-5 yr M	1¾-5 yr F	6-15 yr M	6-15 yr F	16-50 yr M	16-50 yr F	over 50 yr M	over 50 yr F
Trauma							2	2	7	7
Cataract	1			1	2	2	14	9	41	28
Glaucoma						1	3	6	5	5
Keratoconj.					1	1	9	8	8	5
Trachoma					3		14	29	16	15
Keratomal.	5	7	21	19	27	12	12	8	2	1
Variola							4	2	3	3
Leprosy							1		1	
Optic nerve atrophy					3		7	11	3	3
Chorioret.			1		2	1	14	5	2	1
Retinal detachment, tumour, pterygium, vitreous hemaorrhage : monocular cases only.										
Congenital					2		1			
Uncertain					1	1	4	4	3	1
Unknown						1	4	4	3	1
Total	6	7	22	20	41	19	104	92	100	77
of whom were healed by surgery :										
Cataract				1	1	2	14	8	33	20
Entropion					1		2	7	3	2
Iridectomy		2			1	1	4	4		1
Glaucoma						1				
Leaving blind	6	5	22	19	38	15	84	73	64	54
									Total :	381.

Gomperts Atjeh (151).

Cause	Boys	girls	men	women
Trauma			1	
Cataract	1		10	11
Cat. complic.				1
Cat. Morgagni			1	
Iritis sanata			1	
Inflammation		2	5	11
Variola			3	1
Glaucoma			1	
Keratomal.	1			
pterygium				1
Optic nerve atrophy				1
Panophth. post operat.			1	
Blennorrhoea				1
Optic nerve atrophy in childbed				1
Retinit. prolif. post operat.			1	
Degeneratio corneae		1		
Neuritis optica			1	
Undetermined	19	9	21	17
Total	21	12	46	47

Around the same time, in 1937, Dr. Gomperts made a survey in the villages of another part of Sumatra, Atjeh, the northernmost tip of the island. He collected i.a. figures for blindness. I give his list, leaving out the unilateral cases.

In the region of Atjeh Trachoma as well as conj. gonorrhoica are never seen. If this list is compared with that of v. Manen in the same period and in the same island, it becomes certain that it is not possible to draw conclusions on the causes of blindness from an investigation in one region only, because of the great variety in races, geographic and social conditions, even in not very distant parts of the country.

Oey Khoen Lian collected 1945-1948 at Tegal (North Mid Java) and later at Semarang, not far from the first town, resp. 1032 and 1312 blind eyes. It is not clear from his report how these eyes are to be divided over unilateral and bilateral blind persons. (1953. 333) Maäs calculated the most important causes of blindness in percentages from the figures of Oey, as follows: (1960. 250)

	Tegal	Semarang
Trachoma	27%	12%
Gonorrh.	34%	24%
Cataract	17%	17%
Glaucoma	8%	11%
Xerophth.	2%	5%
Iridocycl.	2%	5%
Trauma	1%	3%
Tumour	—	1%

Maäs (l.c.) also quotes an investigation by Gardjito, made in 1939-1940 of which I could not find the original report. Figures as stated by Maäs, citing Gardjito:

	Trach.	Gonorrh.	Xerophth.	Catar.
Kendal	23.7%	45.24%	—	6.6%
Pemalang	39.8%	22. 8%	17.6%	10.4%
Pekalongan	48.7%	24. 8%	4.3%	2.9%

All towns are situated on the north coast of Mid Java, and percentages for conj. gonorrhoica are extremely high. It is not clear in which way the diagnosis was made. Oey explains his high figures as one of the sequelae of war and revolution, but Gardjito's high figures were prewar.

In 1957 during a WHO sponsored trachoma pilot project in Mid Java in 9 villages around Semarang the number of patients blind by various causes was stated as follows:

Vision impaired by trachoma	34 persons blind	
Idem by conjunctivitis and/or nutritional disease	53 ,, ,,	Total examined 14,996.

Maäs (1960.250) gave a report on the number of blind persons seen at Medan Eye Dept. of the Faculty of Medicine of the University. Among 7025 patients seen during 1959 he found 1181 blind eyes. Limit of visual acuity: 2/60. His list, which mixes unilateral and bilateral cases is given as follows :

Cause	M	F	total		M	F	total
Vulnus bulbi	12%	6%	10%	Uveitis	2%	3%	3%
Contusio	4%	0.5%	3%	Glaucoma	2%	5%	3%
Phthisis Maculae Staphyloma	6%	6%	6%	Optic nerve atrophy	1%	0.5%	1%
				Retinitis	1%	1 %	1 %
Cataract	58%	61 %	59%	Conj. Gon.	—	1%	0.5%
Other congenital	5%	6%	6%	Retinal detachment	—	—	— (3 male)
Trachoma	1%	4%	4%	Retinobl.	1%	1%	1%
Other diseases	5%	5%	5%	Other tumour	1%	0.5%	1%

Total 717 eyes of male persons 467 eyes of female persons, 1184 eyes

Comment of Maäs: Patients seen at outpat. dept. : 4052 male ; 2973 female. During 1959 7.7% of outpatients had Trachoma, 1% had Xerophthalmia, only in the first stage without corneal involvement. Comparing his figures with those found in Mid Java by Gardjito and Oey he concludes that every region needs its own type of measures for prevention of blindness.

At Bandung during not quite one year's time, medio 1958 — medio 1959, all blind patients seen at the Outpat. Dept. of the Tjitjendo Eye Hospital were classified by Mrs. A.A. Seniman (Go-Sennema) (150). She divides the patients in a group where the cause in both eyes was the same, 468 patients, and another group, in which there was a different cause of blindness in the two eyes, 125 patients.

Cause of blindness, all ages, 468 patients, same cause in both eyes.

Leucoma corneae	101	21.5%
Keratomalacia	86	18.3%
Trachoma	81	17.3%
Cataract	48	10.2%
Optic nerve atrophy	32	6.8%
Glaucoma	20	4.2%
Iritis, iridocycl. uveitis	17	3.4%
Tapetoretinal degen. etc	15	3.2%
Phthisis bulbi	13	2.7%
Ulcerative keratitis	10	2.1%
Other causes	45	9.6%

Of children under 10 years of age with the same cause of blindness on both eyes, the figures follow :

Keratomalacia	84	58 %
Leucoma corneae	33	22.7%
Phthisis bulbi	5	3.4%
Cataract (not operated)	4	2.7%
Congenital anomalies	4	2.7%
Lesions of optic pathways & visual centres	4	2.7%
Ulcerative keratitis	3	2 %
Trachoma	2	1.3%
Conj. Gonorrh.	2	1.3%
Papiledema by intracran. neoplasm	1	0.6%
Retinitis	1	0.6%
Neoplasm of retina	1	0.6%
Tapetoretinal degener.	1	0.6%
Total	145	

Of 125 patients with different causes of blindness in the two eyes, 183 eyes were blind finally (after treatment, insufficient treatment or after refusal of treatment). Causes of blindness in 183 **eyes** (not persons) were as follows :

Phthisis bulbi	65	35.5%
Leucoma corneae	58	31.6%
Trachoma	14	7.6%
Iritis, iridocycl. uveit.	14	7.6%
Cataract	6	3.2%
Glaucoma	5	2.7%
Other causes	21	11.4%
Total	183 eyes.	

From a more detailed table, giving also the age groups, it can be seen that in the age groups 20—39 yrs. there is one man and two women blind by conj. gonorrh. as well as 2 girls. This figure is much lower than that seen in Surabaja, but otherwise the percentages of Bandung and Surabaja are not essentially different, even unto the high figure for blindness by Keratomalacia.

Serological reactions :		pos.	neg.	??
Leucoma corneae	101	9	15	77
Optic nerve atrophy	32	14	18	
Iritis etc.	17	3	9	5
Ulcerative keratit.	10	1	—	9
Chorioretinitis	6	1	4	1

The most recent figures published were those of Mrs. Tamin-Radjamin and Ngurah Oka for the outpat. dept. of the medical faculty at Surabaja (1966 498) for 1962 and 1963. The next is a short rearrangement of their results :

age	0 — 15 years		16 — 40 years		over 40 years	
year	1962	1963	1962	1963	1962	1963
total	15	43	100	112	88	101
Gonorrhoea	20%	9.3%	5%	9%	—	4%
Syphilis	—	—	4%	3%	3.4%	5%
Trachoma	6.7%	—	31%	21%	26%	29%
Tuberculosis	6.7%	—	—	—	1%	—
Meningoc. inf.	—	—	—	—	—	1%
Other infection	—	—	2%	—	1%	—
Trauma	—	2%	—	—	—	2%
Diabetes	—	—	—	0.9%	—	—
Vascular disease	—	—	1%	0.9%	—	2%
Nephritis	—	—	1%	2%	—	—
Neurol. disease	—	7%	—	2%	—	—
Keratomalacia	20%	58%	—	—	—	—
Genetic origin	—	—	1%	5%	—	—
Unknown to science	—	—	5%	10%	18 %	14%
Insuff. evidence	33%	20%	35%	18%	38%	12%
Unknown	21%	2%	14%	28%	11%	25%
Glaucoma	—	—	4%	10%	19%	14%
Senile cataract	—	—	5%	0.9%	—	4%
Optic atrophy	13.5%	—	10%	11.6%	11.5%	8%
(idem syhil.)	—	—	3%	2.7%	2.4%	3%

These percentages are fairly in agreement with those in the present study but probably because of the rather lower totals, especially in children, the difference between '62 & '63 in Keratomalacia is improbably high.

Chapter 15 Comparison between data on blindness in various countries.

In the WHO Epidemiological and vital statistics Report, Vol. 19 no. 9, 1966, figures on blindness are given, collected from various sources. An attempt has been made to organize the figures in a system which makes them more or less comparable. Unfortunately the list does not contain figures on the causes of blindness in Indonesia's nearest neighbors, Singapore, Malaysia and the Philippines, but from other countries the data can be compared to those in the present study.

The figures which are given for Indonesia, originally published by Oey Khoen Lian and Maäs concern a mixture of bilateral and unilateral blindness, which is not clearly stated in WHO's lists (P. 484). For comparison I chose a few African countries, some Asian neighbors, Australia and New Guinea and some European curiosities. I selected the surveys which seemed most trustworthy to me, and avoided those which mix bilateral and unilateral blindness. The countries I chose I gave a Roman number, to make it possible to place them in a table for comparison. The explanation which is necessary is as follows :

AFRIKA.

Cameroon I. Survey carried out on behalf of the British Empire Society for the Blind. 1952. Difinition of blindness : inability to count fingers at one metre distance. Incidence rate : over 1800 per 100.000 inhabitants.

Ghana II. Survey carried out on behalf of the Brit. Emp. Soc. for the Blind. 1952. Definition of blindness: inability to count fingers at one metre. Incidence rate : 3000 blind per 100.000 in habitants.

Kenya. III. Investigation on behalf of the Brit. Emp. Soc. for the Blind, in cooperation with local medical authorities. 1953 & 1955. Definition of blindness : Total blindness only. Incidence rate : 1100 blind per 100.000 inhabitants.

North Nigeria. IV. Survey carried out on behalf of the Brit. Emp. Soc. for the Blind. 1952. Definition of blindness : inability to count fingers at 1 metre. Rate of incidence : 1000 blind per 100.000 inhs bitants.

ASIA.

Aden. V. Survey on behalf of the Brit. Emp. Soc. for the Blind, 1953. Definition of blindness not stated. Incidence rate not stated.

Ceylon. VI a. Central Province, 1944. Definition of blindness : not stated.

VI b. Northern Province (Tamil) 1950-1952. Definition of blindness: Snellen 3/60 or less. Incidence rate 1963 : 470 blind per 100.000 inhabitants.

Hongkong VII. a. Blind adults. 1954. Perception of hand movement or less. Incidence rate 1963 : 1.392 blind per 100.000 inhabitants.

VII b. Blind children. Vide a.

VIII a. India. Uttar Pradesh. District Barabanki. Random sample consisting of 1489 families examined. 1961. Definition of blindness : Adults, central visual acuity of less than 6/60, children less than 6/24. Incidence rate, 1962 : 460 blind per 100.000 inhabitants.

VIII b. West Bengal, Calcutta Medical College Hospital. 1946-1948. Blind patients seen at the hospital. Definition of blindness: inability to count fingers at 1 yard. Including cataract not yet operated.

Japan. IX a. Pupils between 5 and 20 years old of 70 of the total of 77 schools for the blind. Definition of blindness : not stated.

Japan IX b. Kyoto prefecture. Certified handicapped persons, visual acuity less than 0,04. 1958. Incidence rate, 1958 : 208 blind per 100.000 inhabitants.

Australia. X. a. Eastern Goldfield, W. Australia. Survey 1954, Definition of blindness not stated.

X b. Kimberley district, W. Australia. Survey 1953. Vide X a.

X c. South West portion of Australia. Survey 1955. Vide X a

X d. Austral. New Guinea and Papua. 1954/5. Vide X a.

X e. Former Dutch New Guinea, figures from Schubert (1964.420). X f. Australia : analysis of forms filled in by blind persons seeking an invalid pension and those completed for statistical purposes only. 1953-1958.

Definition of blindness not stated. Incidence rate 1933 (census) 59 blind per 100.000 inhabitants.

X g. Tasmania. Blind pension applicants. 1953-1958. Definition of blindness : not stated.

XI. Iceland. Survey 1955 of blind persons registered in 1950 census, mostly living at Reykjavik. Definition of blindness : 3/60 or less with best correction. Incidence rate, 1954 : 272 blind per 100.000 inhabitants (many consanguinity cases).

XII. Malta. Systematic search to trace all the blind of Malta and Gozo. 1958. Definition of blindness : less than 3/60, or inability to find one's way about in unfamiliar places, with best correction. Incidence rate : 1960, 194 blind per 100.000 inhabitants.

The listing of the causes of blindness used in WHO's survey is according to classification as used in the International Standard, i.e. classification by etiology and by site.

WHO's listing gives the original figures. To make them more easily comparable I have calculated the percentages from the total blind persons in each separate survey.

For China there are no recent figures, only a rather elaborate list of congenital deformations by Feng Pao Hua (1959.115). Flowers (1968.125) gives a digest of prewar figures for causes of blindness in China (which I add after the figures as published by WHO).

Table 12 Causes of blindness in several countries, according to data from WHO'S Survey, 1966, by etiology and site of affection.

Contry	I	II	III	IV	V	VIa	VIb
total	332	1230	1093	2330	290	1802	543
According to etiology :							
total infect.	64%	80%	5%	65%	5.8%	—	—
Gonorrh.	—	—	—	0.08%	—	—	—
Variola	4.8%	1.6%	—	7%	3%	—	—
Syphilis	6 %	2.6%	—	7%	—	—	—
Trachoma	27.4%	20%	3.6%	30.6%	—	—	—
Onchocerc.	23.5%	45.4%	1.3%	2.4%	—	—	—
Lepra	—	0.6%	—	5.5%	—	—	—
Trauma	1.8%	0.8%	1.5%	1.7%	7.3%	2.7%	1.5%
Tumour	—	—	—	0.2%	0.4%	—	3.7%
Total gen. diseases	0.3%	1.5%	—	1.2%	—	9%	—
Keratomal.	0.3%	1.5%	—	1.1%	—	9%	—
Prenatal etc.	—	—	—	0.2%	—	2%	1.6%
Etiol.??	33%	17.6%	93.4%	31.2%	86.5%	77.3%	93.2%
According to Site of affection :							
Eyeball in general:							
Total	12%	4.7%	22%	7.6%	9.8%	9.5%	13.8%
Glaucoma	6.6%	0.9%	4.5%	2.3%	2.4%	5%	7.1%
Struct. anom.	—	—	—	0.08%	—	—	1.6%
Myopia	5.4%	3.8%	0.5%	5.1%	—	1.5%	4.6%
Conjunctiva	—	—	—	0.04%	—	—	0.5%
Cornea	35.8%	49%	20.3%	52%	32.4%	64.4%	5.3%
Lens	17.9%	7.4%	45.8%	18%	16.2%	28%	59%
Uvea	12%	32.5%	2.1%	12.3%	—	7.2%	1.5%
Retina	10%	3.5%	1.1%	4%	—	1.4%	5.3%
Optic nerve	4.2%	2.2%	5.7%	4.6%	—	3.1%	3.1%
Vitreous	—	—	—	—	—	—	0.15%
Undetermined	8.1%	0.2%	2.4%	1.1%	41.7%	4.4%	10.6%

Countinued. WHO'S Survey 1966, by etiology and site of affection.

Country	VIIa		VIIb		VIIIa	VIIIb	IXa	IXb
Total	800		200		251	2862	7032	3892
	M	F	M	F				
According to etiology:								
Total infectious	19.2%	17.1%	—	—	33%	26 %	16%	10.6%
Gonorrh.	—	—	—	—	1.2%	0.9%	1%	1.2%
Variola	—	—	—	—	8.7%	2.3%	0.1%	—
Syphilis	10.2%	2.6%	—	—	—	6.1%	4.4%	2.9%
Trachoma	9 %	4.5%	—	—	23.5%	5.2%	0.7%	4.6%
Onchocerc	—	—	—	—	—	—	—	—
Lepra	—	—	—	—	—	0.06%	—	—
Trauma	17 %	4.6%	—	—	8.8%	0.6%	4.2%	5.2%
Tumour	—	—	—	—	—	0.18%	0.3%	0.4%
Total general diseases	24%	29%	60%	71%	1.5%	9.5%	8.3%	3.1%
Keratomal.	24%	29%	60%	71%	1.5%	8%	7.7%	1.4%
Prenatal	—	—	17.5%	22%	—	3.3%	56.6%	2.4%
Etiol.?	39.8%	49.3%	22.5%	7%	55.5%	59%	14%	5.6%
According to Site of affection:								
Eyeball in general:								
Total	5.7%	9.5%	—	—	4%	14%	39%	33.4%
Glaucoma	5.7%	9.5%	—	—	4%	9.3%	1.2%	4.7%
Struct. anomalies	—	—	—	—	—	0.6%	21.8%	7%
Myopia	—	—	—	—	—	0.76%	15.6%	21.5%
Conjunctiva	24%	29%	60%	71%	13.5%	0.06%	1.1%	—
Cornea	9%	14.5%	—	—	25.9%	6%	20.4%	22.5%
Lens	20%	30%	9.5%	12%	45.4%	46%	11.9%	7.8%
Uvea	3.5%	2%	—	—	—	1.8%	2.4%	5.3%
Retina	—	—	—	—	—	—	7.2%	11.7%
Opticnerve	1%	1%	6%	5%	—	2.6%	6.6%	13.4%
Vitreous	—	—	—	—	—	—	0.04%	0.1%
Undetermined	37%	14%	24.5%	12%	11.1%	18.8%	10.6%	5.7%

Continued, WHO's Survey, 1966.

Country	Xa	Xb	Xc	Xd	Xe	Xf	Xg
Total	28	149	3	96	85	2806	157
According to Etiology :							
Total infect.	69%	79%	—	21%	—	5.7%	9%
Gonorrhoea	3.6%	2.7%	—	1%	—	0.5%	—
Variola	—	—	—	—	—	—	—
Syphilis	—	—	—	—	—	—	—
Trachoma	66%	27%	33%	20%	4.7%	2.2%	—
Onchocerc.	—	—	—	—	—	—	—
Lepra	—	0.6%	—	—	—	—	—
Trauma	10.7%	9.3%	33%	6%	2.3%	5.6%	7.7%
Tumour	—	—	—	—	—	1%	0.6%
Total general diseases	—	—	—	—	—	15.9%	19%
Keratomalacia	—	—	—	—	present	—	—
Prenatal					1.2%	15.5%	14 %
Etiology.??	21%	13.5%	33%	71%	91%	56.3%	50 %
According to Site of affection : Eyeball in general :							
Total	3.6%	3.3%	33%	14%	—	25.5%	20%
Glaucoma	—	2.7%	—	1%	2.3%	16%	15.3%
Struct. anom.	—	0.6%	—	—	8.3%	—	—
Myopia	3.6%	—	—	12%	—	9.1%	—
Conjunctiva	7%	2.7%	—	1%	—	0.7%	—
Cornea	66%	75%	33%	33%	21%	6.6%	4.4%
Lens	25%	14%	33%	39%	52%	19.8%	24.4%
Uvea	—	—	—	7%	2.3%	4.5%	10.2%
Retina	—	—	—	—	2.3%	21.7%	22.2%
Optic nerve	—	—	—	5%	3.6%	9.2%	17.6%
Vitreous	—	—	—	—	—	0.3%	0.6%
Undetermined	—	4.6%	—	—	8.2%	11.9%	0.6%

Continued, WHO's Survey 1966.

Country	XI	XII	Hawaii
Total	143	638	?
According to etiology :			
Total infect.	—	13.4%	—
Gonorrh.	—	—	—
Variola	—	—	—
Syphilis	—	—	4.5%
Trachoma	—	12.3%	3.2%
Onchocerc.	—	—	—
Lepra	—	1.1%	—
Trauma	2.1%	1.7%	7%
Tumour	—	1.2%	—
Total general diseases	—	15.2%	—
Keratomal.	—	—	—
Prenatal etc.	4.2%	9.2%	7%
Etiol. ??	93.7%	59%	—
According to Site of affection :			
Eyeball in general :			
Total	57.3%	38%	—
Glaucoma	52.5%	17%	6%
Struct. anom.	0.7%	2.2%	—
Myopia	4.2%	18.6%	—
Conjunctiva	—	—	—
Cornea	8.4%	12.3%	—
Lens	8.4%	21.1%	27%
Uvea	2%	2.2%	—
Retina	12.6%	0.6%	10%
Optic nerve	4.2%	7.5%	10%
Vitreous	—	—	—
Undetermined	2.1%	18%	—

For Hawaii I add figures as given by Pinkerton (1956.373). The data which Schubert collected in former Dutch New Guinea, now Irian Barat, I put next to the figures concerning Australian Nw. Guinea in the listing (420).

From some countries I could not find data on causes of blindness, but from Fuchs' "Geography of Eye Diseases" I collected a few figures on the incidence of the most important blinding diseases and compared them with those found at Undaan Eye Hospital. In that way it is possible to get an impression on similarities and dissimilarities in various countries. As far as the incidence of eye diseases goes, it seems that for : Xerophthalmia, Conjunctivitis, gonorrhoica, Trachoma and Glaucoma the figures as seen at Surabaja are very high and as these diseases are prime blinding diseases also, we may expect that they will result in much blindness.

CHINA, Flowers, 1948, compiles data from Cheng, Cunningham, Shen and Ling and gives as his opinion, that important blinding diseases in China have been : ulcerative keratitis of unknown origin (Koch-Weeks, Gonococcus, Pneumococcus most frequently found) Trachoma, especially in adults, Phlyctaenular Keratitis; Keratomalacia; Gonorrhoeal conjunctivitis, more frequent in adults than in the newborn ; Smallpox and measles ; Cataract in older people ; Glaucoma.

Not common are : Syphilis, Trauma, congenital and hereditary affections.

The data are very old, it is quite probable that some of the diseases named have meanwhile got a different importance.

Howard Harvey (1926.182) estimates the number of totally Blind people in China at 500.000, almost blind 5.000.000 and very weak-sighted 15.000.000 persons.

W.P. Ling at Union Medical College at Peking, ca. 1925, examined 1038 blind individuals, of which number the cause of blindness were as follows :

Trachoma	52%	Diverse infections	6½%
Cataract	11%	Keratomalacia	½%
Lues	10%	Other causes	5½%
Glaucoma	9½%		

These figures are cited by Marcel Leger (1931. 229) who adds, that possibly cataract and glaucoma are seen at an early age because of previous Trachoma.

Feng Pao Hua (1959.115) gives a very detailed description of the congenital abnormalities found among 215,366 outpatients : in 0.0647% of the total number of patients there existed a congenital anomaly causing blindness (anophthalmos, optic nerve coloboma, congenital leucoma, congenital leucoma adhaerens, congenital coloboma maculae luteae, hydrophthalmos) whereas in 0.4636% there were anomalies which probably caused a very low visual acuity (microphthalmos, coloboma iridis et chorioideae, cataracta congenita, aniridia).

Shen D.S. (1945. 429) found 1130 blind eyes among ca. 850 patients in 4 years' time in West China, citing the following causes :

Trachoma in 438 eyes
Ulcerative keratitis 158 eyes
Keratomalacia in 51 patients
} 6 adults, the others children.

Chang (1949. 53) reports on 14.982 outpatients of whom 1.037 were blind in one eye only, 963 blind in both eyes (113 with visual acuity 00). He includes patients of whom the prognosis was still "indefinite", which makes the figures useless for comparison with other statistics for causes of blindness, which normally take only into account patients who are definitely and permanently blind.

INDIA. In a comment on the survey in Uttar Pradesh (426) Sharma states that the local incidence of blindness was 1.118/1000,00, i.e. $5^{1}/_{2}\times$ the incidence in England and Wales. There were far more blind women than blind men, because cataract, Trachoma, mucopurulent ophthalmia, variola and glaucoma are far more frequent among women, for which there are various reasons, among them sociological ones. The rate of blindness increases with age, with lower economic levels, and was among : Hindus 50,82/100.000 ; Muslims 55,62 ; Haryans 69,28.

In blind eyes the cause was cataract in 34%
Trachoma 20.64%
Mucopurulent infections 16.76%
Variola 13.79%

Also seen : sequelae of couching operation in cataract, glaucoma, trauma, ophthalmia neonatorum, nutritional deficiency. 38% would have been curable if seen in time, 58% would have been preventable.

NORHT VIETNAM.

Nguyen Dinh Cat (1953. 321) states that Trachoma is solely responsible for 50% of the cases of blindness (specific lesions as well as secondary infections). Glaucoma is seen as frequently as in occidental countries. It is impossible at the time to establish the correct number of blind people in the area.

HONGKONG.

G.C. Dansey-Browning (1958. 73) states that syphilis accounts for 10% of blindness in males. Other blinding diseases which are frequently seen are :

1. Acute infection of the anterior segment of the globe combined with various degrees of subnutrition.
2. Phlyctenular disease.
3. Trachoma.
4. Untreated cataract.
5. Glaucoma.

Of interest is, that nasopharynx tumours, presenting with paralytic strabismus are seen in 50 patients annually. This type of tumour is not rare at all on Java also.

JAPAN

Akira Nakajima (1960. 5) gives an interesting comparison between statistics collected in 3 periods : 988 patients seen between 1910 - 1929,
3,645 in 1952,
8.700 in 1960,

The relative importance of infectious causes of blindness, nutritional causes and other causes slowly decreases during the years, whereas the proportion of congenital anomalies causing blindness increases :

	1910 — '29	1952	1960
infectious eye disease	36.5%	18.8%	11.4%
Congenital anomalies	29.7%	52.3%	72.8%
Nutritional deficiency	15 %	9.6%	6.9%
Other causes	18.8%	20 %	9 %

He compares these figures with those published by Sorsby (1959). It seems that Japan has reached the same low level of preventable and curable blindness as England where also 72.4% of the cases of blindness is caused by congenital anomalies.

NEW GUINEA, now part of Indonesia, named IRIAN BARAT.

Schubert (420) worked in New Guinea as the region was still under Dutch administration (1961-2) and describes at length his experience, especially on Trachoma. He lists binocular causes of blindness seen (I placed this list next to that of Ida Mann among the data collected in WHO's Survey above) and adds a list of monocular causes. Agreeing with Mann's experience in Australian Nw. Guinea and also among Australian natives, Schubert also saw injuries as the prime cause of monocular blindness ; hunting and walking through the bush he states as the reason.

Commenting on the very low number of glaucoma cases and the few cases of mature cataract, whereas there is immature cataract, he makes the point that life expectancy as such is low already, but still more so for blind or other handicapped people. This is especially true for children, but also for older people, who are the victim of the very hard struggle for life under the socio-economic circumstances as they are prevalent in the region.

From his report it is clear that in different regions inside Irian (which is after all an enormous island) there are very different eye diseases and

Table 13

Incidence of some blinding diseases in Outpatients' Departments in various countries. Figures chosen from tables in A. Fuchs : "Geography of Eye Diseases". (132)

Hospital	Total	Xer.	Go.	Trach.	Glauc.	Catar.
S. Tomas University, Manila, Philippines	10355	0.1%	—	0.90%	1.2%	1.2%
General Hospital, Singapore	15795	0.15%	0.01%	4%	0.4%	3.2%
I. General Hospital, Taipeh, Taiwan	5250	—	—	47.7%	0.5%	0.07%
Hospital Nam Viet, Cholon, S. Vietnam	ca. 27500	0.18%	0.06%	18%	0.15%	0.6%
Muslim Free Eye Hospital, Rangoon, Burma	12066	0.3%	0.5%	20.7%	17%	2.3%
Victoria Eye Hospital, Colombo, Ceylon	70479	0.3%	0.01%	0.07%	0.09%	2.1%
Undaan Eye Hospital, Surabaja, Indonesia 1955	13340	1.1%	2.4%	27%	1.1%	1.9%

consequently the relative value of the incidence of blindness and its causes differs, calling for local variation in appropriate measures of prevention.

Comment on the Incidence figures in some neighboring countries :

Trachoma. At the time of comparison (mid-fifties) only Taiwan had a higher incidence of Trachoma, according to the data collected by Fuchs, using questionnaires sent to all known eye hospitals.

Keratomalacia. This figure is highest for Surabaja, but the figures in the same book from Jogja and Bandung are as high.

Conjunctivitis gonorrhoica. Surabaja at the time was one of the busiest harbor cities of S.E. Asia, but it would seem probable that a large harbor like Singapore should have a high incidence for gonorrhoical disease also. In the whole collection of figures only the incidence in Egypt is higher than that at Surabaja.

Glaucoma. Manila has the highest incidence in the neighborhood, but Surabaja is almost as high.

Cataract. Only for the incidence of cataract is Surabaja taking the middle of the road, which supports the common opinion that cataract in Indonesia is not overfrequent.

Phlyctenular conjunctivitis. Ubaldo and Ocampo (1939. 525) state that in the Philippines phlyctenular conjunctivitis belongs to the most important blinding diseases, along with injuries, keratomalacia and retinoblastoma in children. It can be stated that at Surabaja true tuberculous keratoconjunctivitis as a blinding disease is rare : the disease occurs, but most often it only impairs visual acuity somewhat, not very seriously.

AGE AT ONSET OF BLINDNESS.

Because of the difficulties in history taking it was not possible to ascertain the age at onset of blindness in many of the patients in the present study. Certain conclusions can be drawn from the division according to age groups, but in the older patients it is quite possible that they have been blind for years or even decades at the time they were examined. We noted the age at the time of examination as best as we could, but that is the only investigation we could make with any degree of trustworthiness.

In the survey published by WHO 1966 (568) for a few countries tables are given for the age at onset of blindness (Table 4, p. 466 - 469) from which I chose a characteristic one for each continent :

I. Survey by Doret, Canton Geneve, Switzerland, 1950. Total 245. Definition of blindness : inability to count fingers at more than 1 metre. Incidence rate, all ages, census 1910 (!): 58/100.000.

II. Survey at Barabanki, 1961, India, Uttar Pradesh. Total 422. Definition of blindness : adults less than 6/60, Children less than 6/24. Incidence rate, 1962, 460.

III. North Nigeria, Census 1944. Total 254. Definition of blindness : not stated. Incidence rate, 1953 : 1000 per 100.000 inhabitants.

Table 14 Age at onset of blindness for Geneva, Barabanki and N. Nigeria.

Age	Geneve	Barabanki	N. Nigeria
at birth	14		
0— 1 yr.		8	
0— 9 yr.	8	63	
0— 5 yr.			61
6—10 yr.			34
10—19 yr.	9	56	61
20—29 yr.	18	37	27—156
30—39 yr.	17	25	24
40—49 yr.	22	70—189	14
50—59 yr.	30	75	17
60—69 yr.	56—118	57	13
70—79 yr.	53	31	3
80— hegher	18	—	—
Total	245	422	244

Comment : If we calculate the point at which half of the patients are already blind, we see that at age 20 in Nigeria 50% of the people who will be blind are already blind; this point is reached in India at 40 yr., and in Switzerland at 60 yrs. According to Sorsby, 1966, at the end of 1962 of the new registrations in the blind register 86.1% was over 60 years of age!!

From Table 1 giving the age at examination in the present study, it can be calculated that ca. 50% of our blind patients is under 40 yrs. old. This places East Java in a group with India, which does agree with other indexes. In countries which have a long tradition of ophthalmology and where the life expectancy is high, this halfway point will be reached at a later age, than in countries where ophthalmology is relatively new in its effect and where life expectancy is still low.

CHAPTER 16. CONJUNCTIVITIS GONORRHOICA.

That conjunctivitis gonnorrhoica has been a blinding disease of importance in this region has been known for a long time. Steiner (1894 480) found among 660 blind persons in Surabaya 123 (18.65%) who were blinded by this cause and calculated that 1 pro mille of the native population was blind at that time from conjuncivitis gonorrhoica.

Not only in large seaports, like Surabaja, but also in agricultural communities the disease was found, as is stated by Zegers Verhoeven (1912) from Modjowarno, were 20 cases were seen annually. Westhoff (1911 563) states that 50% of the inmates of the Bandung Institute for the Blind (founded in 1901) was blinded by this cause. In view of the fact that in old cases it is often difficult to differentiate between the sequelae of conjunctivitis gonorrhoica and of Keratomalacia, a disease which Westhoff describes in the same paper, it is possible that part of the 50% in fact were caused by the latter disease, but probably the number remains high, as many of the children blinded by Keratomalacia die young and conjunctivitis gonorrhoica is not so dangerous for life and in this country it is not especially a disease of babies and children, but of all ages. In the older literature from Europe similarly high frequencies are quoted for the inmates of schools for the blind. Sorsby (1946. 469) mentions that in 1884 ophthalmia neonatorum was found to be the responsible cause for the blindness of from 30 to 41% of theinm ates of selected Blind Institutions in England. In his recent Comment on the WHO Survey on Causes of Blindness (1967. 473) he qualifies the finding of high figures for conjunctivitis gonorrhoica in underdeveloped countries now and in the older literature from Europe, in that he says, that not all ophthalmia neonatorum is caused by the gonococcus. This is true, but other forms of ophthalmia neonatorum are not so dangerous for the eyesight, e.g. inclusion blennorrhoea does not damage the cornea, and other types of purulent ophthalmia in the newborn must be very rare. In this country conjunctivitis gonorrhoica is not synonymous with ophthalmia neonatorum, on the contrary the majority of cases are in older children and adults. This is stated also by Bakker in his chapter In the handbook of Schieck-Bruckner on Tropical Eye diseases (1932.18), and by Mulock Houwer (307) who saw 100-150 cases annually in the Eye Civil Hospital at Djakarta in prewar years.

The route of infection is not necessarily venereal, it can also be transmitted by contact, e.g. dirty fingers. Especially the slendang, a piece of cloth worn by women over one shoulder and used for carrying the baby, for bringing home the purchases from the market, and for wiping the face of all members of the family, is an easy route for transmission of various infectious eye diseases, among them conjunctivitis gonorrhoica and Trachoma, probably often in combination. The habit of all children of the family to sleep on the same bank, using one another's pillows indiscriminately is another source of danger.

Oey Khoen Lian (1953.333) found that conjunctivitis gonorrhoica was the main cause of blindness in North Mid Java, resp. 34% at Tegal and 24% at Semarang. Gardjito found even higher fugures in the same region in '39-'40 (cited Maäs ('60.250). Oey ascribes his high figures to the aftermath of war and revolution, but Gardjito's figures are prewar. The towns concerned are all of them seaports. Medan, which is also a seaport, according to Maäs (1960.250) on the other hand has very few cases of blindness caused by conjunctivitis gonorrhoica, as is in agreement with other findings from Sumatra (Gomperts, 1940,151, van Manen 1938,262).

From the annual reports of the government's Eye Health Service for 1958, 1959 and 1961 it can be seen, that Djakarta and Surabaja have the highest frequency for conjunctivitis gonorrhoica, but also inland cities like Jogja, Bandung and Malang on Java are not free. Seaports on other islands (Palembang, Makasar, Pontianak, Bandjarmasin etc.) have very low figures. Probably there are local differences in customs (adat) in connection with general hygiene as well as with prostitution which must be taken into account to explain these striking differences.

At the Eye Hospital Undaan, Surabaja, in the years covered by the present study there appeared to be a slow decrease in frequency of conjuctivitis gonorrhoica, as seen among outpatients.

1958	1.2% of adult outpatients had conj. gon.	2.4% of children.
1959	1.2%	2.5%
1960	1.2%	2.4%
1961	1.3%	1.5%
1962	0.8%	0.9%

The disease was seen in all age groups, the oldest patient was over 70 years of age. The figures for different ages were for 1962 :

newborn : 18
between 2 and 12 months : 10 } healed without corneal acars.
1-6 years : 19 (5 of them from outside Surabaja). All of them had been sick from 1 to 10 days, and 14 eyes developed corneal complications, one eye was lost).
over 16 years of age : 98.

The cases of blindness by conjunctivitis gonorrhoica in 1962 (3 children and 56 adults) were not all of them fresh cases.

Therapy: In prewar times the care of patients with conjunctivitis gonorrhoica was laborious and hazardous. During the thirties in this country there was a long discussion berween Deutman at Surabaja and several other oculists about the treatment of this disease.

Although it was at that time generally considered malpracitice to cover eyes suffering from purulent conjuctivitis, Deutman applied Priesnitz compresses with Argyrol 20% ointment, and abolished the washing with permanganas kalicus every half hour. This simplified the nursing and reduced the risk of damaging the cornea by the washings, also giving the patient a chance for sleeping through the night. The results were satisfatory especially if fever therapy was added. He had some followers among the other oculists, but in the middle of the discussion the discovery of the sulfadrugs changed everything abruptly. It soon became clear that these drugs worked miracles, and that e.g. babies could be cured by giving sulfa to the mother. In postwar times, as penicillin became available, as well for eye drops as in ointment and injections things looked still better. Eye drops were applied every 2 minutes, for half an hour, after which there were no longer found gonococci in a conjuntival smear. To prevent recurrences it was soon found that the probable source, i.e. the urethritis must be cured also, i.e. injections had to be added. In 1951 at Undaan 192 patients were treated with intramusc. Penicillin 3 × 50.000 U. stopping the corneal complications from increasing. It was often not even necessary to admit the patients, they could be treated as outpatients. Possibly by the use of sulfa drugs and Penicillin on a large scale the incidence of conjunctivitis gonorhoica according to the figures of Salim (1959.406) showed a decrease as compared to former times at Djakarta: 1953 0.19%; 1954: 0.24% 1955: 0.32% 1956: 0.15% 1957: 0.18% (outpatients). The figures for Undaan, Surabaja, have been given before, and showed a decline too.

Unfortunately meanwhile certain strains of gonococci have developped a resistance first against sulfa drugs, later on also against Penicillin. This phenomenon has been seen all over the world (WHO Report on gonococcal Disease 566) and it is our experience that we need much higher doses of Penicillin now than we did formerly (600.000 U to start with) and see more cases which cannot easily be cured, except with very high doses (c.a. 1.000.000 U —) or with other kinds of antibiotics. Also the trend for conjunctivitis gonorrhoica to decrease which was seen until 1962, has not continued: in 1966 0.7% of adult outpatients were suffering from it and 1.4% of children outpatients. The WHO Report even recommends 2.4

mega units of repository Penicillin, and advises combination with sulfa drugs, or with other antibiotics, in an effort to prevent the emergence of resistant strains of gonococci. In this country the indiscriminate use of sulfadrugs and Penicillin in cases where there is no indication for it, and also by unauthorized personnel, like nurses and midwives, who in villages and also in the city treat patients without medical supervision, often using a far too low dosage scheme into the bargain, is probably one of the factors which caused the emergence of resistant strains, as is also stated in the WHO Report.

Prevention. Ophthalmia neonatorum. The classical method of preventing ophthalmia neonatorum is the method of Credé, using Nitras argenti eye drops, 1-2% applied as soon after birth as possible. In countries where this method has been used consistently, the disease has disappeared completely. There are a few drawbacks in the methed: 1. sometimes it is found, especially in hot climates that the strength will increase with time, which makes it dangerous for the cornea (Te Bek Siang 1960 503). There should be stringent orders for midwives: that they are not to use strong solutions of nitras argenti for treating the umbilicus, but use lapis pencil for that, keeping the weak nitras argenti solution for the eyes. This solution should conscientiously be made up every week, not just filling the old bottle up, but cleansing it thoroughly to remove residues. The second danger is the interchange of the weak solution with the stronger one, which can only be prevented by discarding the stronger one altogether (Sie Boen Lian 441.442) Te Bek Siang and Tan Thiam Hok (1960 503) experimentally proved the advantage of nitras argenti over antibiotics, as it is bactericidal as well as an adstringent, whereas antibiotics are bacteriostatic only. This agrees with the experiences in Denmark and New York hospitals, cited in the WHO Report on gonococcal disease : in trials the method of Credé was abandoned in certain wards and replaced by antibiotics with the result that the wards where Credé's method was continued, remained free from cases of ophthalmia neonatorum, whereas some cases were seen in the wards where antibiotics were used (566).

Conjunctivitis gonorrhoica in adults. The first method to prevent this is raising the general hygienic level. In more advanced countries the disease is extremely rare in adults, although this cannot be said of gonorrhoea generally. The more important method of prevention is of course the eradication of gonorrhoea as such. Following the optimism in the early prewar years it now becomes clear, that gonorrhoea is not yet finished, because of several factor which make its eradication difficult : the short incubation period makes it more difficult to trace contacts after the infectious exposure, the detection of the disease in the female reservoir is troublesome, and all important : the emerging resistance of certain strains of gonococci makes therapy uncertain again.

The report of WHO on gonococcal disease rightly recommends well planned and well implemented campaigns for eradication of this still dangerous disease. If an incentive were needed for this undertaking in this country the figures in this study could be used, to illustrate the very serious damage which is still to be ascribed to this menace. We have not yet reached the stage as in Vienna, where Safar (1962) could say that a demonstration case of adult conjunctivitis gonorrhoica was the first one he saw in 30 years.

Chapter Seventeen.

SYPHILIS.

As a problem in ophthalmology Syphilis has nearly vanished from the world literature. Whereas before 1930 there were still numerous papers published on syphilitic eye disease, afterwards cases have become rarer and rarer, interest has been lost, until now in European and American literature there is scarcely anything to be found on the subject. In Indonesia, especially on Java, things are quite different. Sumatra with a population with quite another kind of adat (traditional customs) has always had very little venereal disease generally as is stated by all specialists who have worked there as well as by the oculists (Maäs 1960. 250 Gomperts 1940. 151) but on Java from early times on there has been much syphilitic disease, also eye disease.

In this country the diagnosis of Syphilis is somewhat complicated by the necessity to differentiate it from Yaws, which formerly was very widespread also. The general rule-of-thumb: Syphilis is a disease of the city, Yaws is a rural disease, in this form is not valid here: Syphilis also is found in rural areas (Dake 1936. 70.), although Yaws generally is limited to villagers only. After the mass campaigns against Yaws in the fifties on Java have been a great success, until there even are not found any fresh cases any more for teaching medical students, the only difficulty in the differentiation which remains is that Yaws after complete healing can retain positive serological reactions, with a high titre even; thus positive serological reactions on Java certainly does not mean Syphilis; they maybe are caused by healed Yaws.

The Eye Hospital Undaan has been fortunate in that we have had the opportunity to cooperate intensively with the VD Research Institute, where from 1955 until 1964 all our patients who had positive serelogical tests were sent for diagnosis, except those who did not return after the first examination of course. For that reason the diagnosis Syphilis as presented in this study is not made by an oculist but by specialist-venereologists. We are grateful for the very useful cooperation with our colleagues from the Institute, who often performed examinations of the liquer cerebrospinalis also.

In those years often several hundreds of patients were sent there by us, and returned to us with advice on treatment. This consisted in Penicillin (PAM) injections, 4.8 million Units, for neurosyphilis 12 millions, if necessary repeated after half a year. We were provided with PAM by the Institute, and even in times of general scarcity we never had difficulty in obtaining it. As a sample of the frequency of cases with positive serology I give the numbers for 1962 : 197 patients with Syphilis, 124 with Syphilis and/or Framboesia, and 29 with Framboesia, 350 patients in all we sent to the VD Institute. In 1966 we had 190 patients with positive serological tests, in 1967 still less : 120. Generally we ran serological tests on all patients with optic nerve atrophy, all forms of uveitis and many cases of corneal disease, which did not look quite simple. In agreement with personal communications from colleagues from the Institute we have the impression that Syphilis is decreasing in frequency. There have been several types of action in the fight against Syphilis, and possibly also the Yaws campaign has had some favorable effect on the Syphilis frequency too,

Syphilitic eye disease in Indonesia.

There exists a curieus discrepancy between what "everybody believed" and what oculists saw. In the thirties it was discovered that congenital Syphilis which had been thought non-exixtent on Java, in spite of the known high frequency of Syphilis in adults, was not so rare after all (de Jong-Martis 1938, 192), and that Tabes dorsalis, which also had been thought to be non-existent, was to be found if looked for. All the time from the statistics of Steiner until those of Westhoff and others, the diagnosis keratitis parenchymatosa and primary optic nerve atrophy had been made and published. The belief, that congenital Syphilis did not exist, was possibly caused (de Jong-Martis) by the fact that many of these children died very early, often without medical help. Keratitis parenchymatosa, which usually begins around the age 6—10 years often is only treated by the oculists, with the consequence that pediatricians are not aware of its existence. Steiner (1896. 479.) saw 6 cases in 3104 patients, Westhoff saw in 1909, 1910 and 1911 resp. 16 in 2536 patients, 27 in 2901 patients, and 24 in 2710 patients (562, 562a, 564).

Kirschner (1926. 205) wasone of the first who published 3 cases of Tabes dorsalis in Indonesians. At that time, 1926, theories about the rarity of Neurosyphilis among "primitive" people were stil prevalent, along with explanations which accused such dangers as : civilisation, alcohol, salvarsan, "surménage intellectuel" and other myths as the cause of Neurosyphilis in "civilised" countries. Uhthoff's opinion (1911. 526) that all cases of genuine primary optic atrophy are really cases of Tabes dorsalis had apparently not been accepted, otherwise the cases seen by Steiner (38 in 1896) and Westhoff (12, 18 and 27 resp. in 1909, 1910 and 1911) would have been interpreted as Tabes.

In Steiner's time Wassermann's test had not yet been found (1906) and it is not clear from Westhoff's reports whether at that time the test was already in use at Bandung.

After the breakthrough caused by Kirschner's paper more and more cases of Tabes dorsalis were described. Van Wulfften Palthe, professor of Neurology at Djakarta in the thirties, wrote a series of articles on the subject (583. 585. 586. 587) in which he drew attention to a few characteristics of Tabes as seen in this island, especially the fact that optic atrophy often is an early sign. Contrary to the old dictum that the disease will stop after having reached the optic nerve, he stated that here it will progress after that also. Sie Boen Lian (437) wrote a paper in 1936, reporting on 35 cases of Tabes dorsalis seen at Djakarta medical school (including cases from the neurological dept.) in 8 years' time, and analyzed the eye signs. Optic atrophy was quite frequent, as well as various pupillary signs. Van Wulfften Palthe saw in the following 5 years 84 cases, again with the same high frequency of optic atrophy (1941, 586) i.e. in 56% of all Tabes cases, whereas the highest figures for Europe at the time was 21.5%.

Others (Zuidema, Jogja 603) Buning (Malang 46) have given case presentations and Bieger (1940, 25) even describes a case in a Batak man, in spite of the rare occurrence of Syphilis on Sumatra.

Other eye diseases caused by Syphilis which were described comprise: Keratitia pustuliformis profunda (Fuchs) by Mulock Houwer, 1935 (296) ; iritis luetica (Soemitro Hadibroto 1936, 459, Mingelen, 1936, 285 Mulock Houwer 1936, 298).

Bakker in the Handbook of Schieck-Brueckner 1932, 18) had stated that 90% of iritis cases seen at Djakarta were caused by Syphilis. Mulock Houwer described a case of Syphilis orbitae (1936, 302) and Steenhuisen (1935, 478) saw a case of gumma of the optic nerve which regressed after specific therapy. Wyono (1941, 591) confirmed the findings of de Jong-Martis on congenital Syphilis for Surabaja.

From the postwar investigations on causes of blindness that of Mrs Go-Sennema (Bandung 150) reports the number of cases were serological tests were performed, which shows that Syphilis as a cause of uveitis and optic nerve atrophy was certainly not rare at the time. Maäs is explicit (1960, 250) in stating that Syphilis is very rare on Sumatra, whereas the data of Oey Khoen Lian do not allow any conclusion on Syphilis as an etiological factor.

Syphilitic eye disease in East Java.

As Sourabaya is rather a large harbor city it can be expected that there is a high percentage of Syphilis among its inhabitants. In 1957 there were 7.000 registered prostitutes in town, among a population of 1.250.000 (Soetopo. 463). At that time the prevalence of Syphilis among some groups of the population which had been investigated, was 15 — 20%. Not only the Syphilis rate is high, but opportunity for treatment is easy also. Not so in rural districts: therefore it is not astonishing that many of our cases of Tabes dorsalis come from distant villages and only in the stage where they have already nearly or completely gone blind by optic nerve atrophy.

Of all cases of uveitis (including iritis, irodocyclitis and choroiditis, acute, chronic and healed) which we saw in 1962 : 2 were caused by a fever, 2 were sympathetic ophthalmia, 8 were caused by trauma. The rest, 470 cases, had positive serological tests in 172 cases (tests run in 435 cases, not done in 35 cases). One case with negative serological tests, but clinically suspect, was by the VD Institute diagnosed as Syphilis all the same. Of the total of 470 uveitis cases, 20% had Syphilis, 14% had Syphilis and/or Yaws, 65% had no venereal disease. This figure is certainly much lower than that of 90% as quoted by Bakker in 1932. Salim (1959, 406) saw 50% Syphilis in uveitis cases at Jakarta.

In 1962 we saw 53 men and 21 women with tabetic optic nerve atrophy of whom resp. 41 and 16 were or went blind. This figure is much higher than that found at Djakarta in the thirties. A possible explanation is, that we now see the sequelae of the Japanese occupation; at that time Syphilis often could not be treated, for many different reasons, and the people who contracted their disease at that time are the tabetics of the years around 1960 : Tabes arising some 12 — 16 years after infection.

From the tables in the chapters on classified causes of blindness it can be seen, that at least in and around Surabaja Syphilis is still an important blinding disease. This fact cannot be generalized for the whole of Indonesia, but calls for local measures of treatment and prevention. This is not the job of the oculist, but we are obliged to call attention to the problem, even if we know that the solution will be very complicated and difficult.

There is a certain "trend" to be seen if we compare the figures in the years 1958 — 62 with 1966/7. There is an decline in the figures for all groups except the middle age group in men :

Year	1958	%	1959	%	1960	%	1961	%	1962	%	1966/7	%
male, 16-40	284		246		214		205		190		106	
Syphilis	36	13%	36	14%	30	14%	30	18%	14	7%	17	total 16%
women, 16-40	232		231		223		128		187		111	
Syphilis	17	7%	21	9%	22	10%	25	11%	19	10%	8	total 7 %
Men, over 40	178		159		189		204		212		120	
Syphilis	26	15%	23	15%	27	14%	22	12%	33	16%	13	total 10 %
Women, over 40	160		198		214		208		211		111	
Syphilis	10	6%	19	10%	11	5%	8	4%	9	4%	5	total 4.5%

Theraphy : In the cases of corneal and uveal Syphilis we often give local corticosteroids besides the specific antisyphilitic treatment. For optic atrophy we add vitamin B1 injections. Keratitis parenchymatosa mostly heals quickly with corticosteroids, although the disease in the second eye seems to be inevitable with whatever kind of treatment. Optic atrophy often has already progressed over the borderline of 5/10 visual acuity ; cases which can be arrested after having deteriorated so far are very rare. Sometimes our Tabetics will show other neurological/psychiatric conditions, which makes it impossible to keep them in the hospital and we have to transfer them to a psychiatric ward, if there is room. These people come often from so far away that treatment in the outpatients' dept. is not possible.

Prevention. If incentives for efforts in VD control were needed the results of our study could be used, as also of gonorrhoea figures. But the complex of socio-economic conditions at the present time defies endeavours in this derection, however much VD specialists and others interested in the field would like to take decisive action.

Chapter Eighteen.

TRACHOMA.

More than any other eye disease Trachoma is feared by the general public in this country, and there is no eye disease about which there is more false knowledge among the semi-educated. After decades of health education in school and press, everybody now knows for certain, that slight redness and itching of the eyes is synonymous with "Trahoom" and will scarcely believe the doctor who says that there is no trace of Trachoma present. At the present time there is no real reason for this phobia, as serious cases of Trachoma among this group of the population grow rarer and rarer each passing year.

After the thesis of Heath (1963, 170) and the report on the Pilot Project on Trachoma of Delon c.s. (80, 81) between 1956 and 1959, there is not much more to be said on the subject. Heath gives the literature in extenso and also an outline for possible treatment and prevention.

Trachoma probably has been found in certain areas of this country for many years. As far as is known Abrahamsz (1875, 3) published the first report on it in 1875 from Borneo (Kalimantan), soon afterwards followed by Steiner (1896, 479) from Sourabaya in the nineties, and Westhoff from Bandung in the first decades of this century, Soewarno from Sourabaya, 1919 (464), and many others after them.

Steiner saw 952 cases of Trachoma among 3.104 outpatients between 1892-5 and 172 cases of entropion (1896, 479). In the Missionary hospital of Modjowarno (100) 983 Trachoma patients were seen in 1908(Duymaer) and 92 cases of entropion among 1260 outpatients. Westhoff's figures for 1909, 1910 and 1911 were resp. : 336 Trachoma, 40 entropion and 11 Trichiasis cases on a total of 2536 ; 359 Trachoma, 61 Entropion, 22 Trichiasis on a total of 2901, and 528 Trachoma, 83 Entropion, 32 Trichiasis on 3730 patients. The importance of the disease at that time was realized not only by the few oculists who were then active, but various health authorities, organized in the Civil Medical Service, gave attention to the problem;

special eye nurses were trained (called dukun mata) by Westhoff at Bandung, and divided over the island of Java according to the needs. For curiosity's sake I give a number of passages from the Annual Report of the Civil Medical Service for the year 1905 (57) relating to East Java :

p.733. Res. Sourabaya, Sidohardjo : Many cases of Trachoma......

p.734. Lamongan...... most frequent diseases are Yaws, Syphilis and Trachoma........

Sedajoe : Yaws and Trachoma are frequent, but patients seldom come for help.

p.735. Res. Pasoeroean : Eye disease, especially trachoma, remains frequent.

Bangil :......Trachoma is still rampant........

p.736. Probolinggo : Granular eye inflammation is seen in moderate frequency.

p.737. Res. Besoeki, Besoeki : ...... Trachoma is not often seen.

p.738. Banjoewangi : Trachoma is very frequent ; the water supply is inadequate ....

p.739. Res. Kediri, Kediri : ...... The number of eye patients, that came for help, was for the town of Kediri 235, from outside the town came 342........

Blitar : Conjunctivitides (catarrhal and Blennorrhoea) were very frequent among the inhabitants, but very few came asking for help, as also Trachoma, which is also frequent. It could be wished that there would be installed a dukun mata, who possibly as a compatriot would be able to win the confidence of the population and could urge them to seek help for their ailments. At present the belief among these patients is very strong, that their eyes will be removed at treatment, and that anyway they will be cut......

p.740. Ngandjoek ......Eye disease is very frequent, especially Trachoma, by which many patients were blind..

Kertosono ......Trachoma is rampant and destroys many people's eyesight......

p.741. Trenggalek......Skin- and eye diseases are frequent......

Paree : ......Eye diseases are still very common and many victims go blind.........

In 1906 there were 21 dukun mata stationed in : Madioen, Patjitan, Pasoeroean, Kediri, Lamongan and Banjoewangi.

Year after year the reports run on in the same vein. Meanwhile Java was the scene of one of the most important events in the history of Trachoma, i.e. the discovery by Halberstaedter and Prowazek of the inclusion bodies, which now are accepted as the cause of the disease (published in 1907, 163). Facilities at Djakarta at that time were clearly already sufficient for this type of scientific work ; probably important discoveries could still be made with simple equipment, without enormous laboratories (Greeff. 152. Soewarno. 464.)

In 1911 Kiewiet de Jonge (204) published an investigation among 700 schoolchildren at Batavia, 6 European children, 133 Indonesian, the rest all kinds of racial mixtures. 24.5% had Trachoma, of which 46% was "acute" and 54% "Chronic", with very few complications!

The history of the various studies of the incidence of Trachoma in different regions of Indonesia can be read in Heath's thesis. Some curious facts : although Trachoma was found in other parts of Sumatra, Gomperts (1940, 151) states that Atjeh at the island's northernmost tip, is free from it. Mulock Houwer (300) draws attention to the fact, that Trachoma in children is relatively benign, but warns against optimism. He feels that part of the Trachoma cases in children will heal but that another part will develop dangerous complications at an older age, if left untreated. School treatment was the method of choice, based on the idea that people who did not contract Trachoma at an early age later on will stay free, and that healed Trachoma cases will not be in danger of reinfection. There is no foundation in fact, as far as I know for these assumptions. As an example the endemic Trachoma in a small part of Amsterdam, eradicated by school treatment, is given, but Mulock Houwer omits to mention that this was supplemented by treatment of contactpersons in the families of the Trachoma patients (Wibaut 571). In my opinion this was the decisive factor in the success at Amsterdam.

The whole picture of treatment of Trachoma, which in these years was based on the painful expression of granules and some adstringent like silver nitrate or copper sulfate, and which took months of patience on the side of patients and doctor, was abruptly changed with the discovery in 1937 by Heinemann (172) of the effectiveness of low dosage of sulfanilamide (Prontosil) per os. He gave a case presentation of 3 quickly successful cases, soon followed by others (Schwarz 1939, 421. Dik 1938, 88. Sie Boen Lian 1938, 439) as was the case all over the world, with the curious exception of Egypt, where the 1937 Report from Giza stated that Prontosil is useless in Trachoma, also in cases with active corneal inflammation (47). Afterwards opinion of course has changed.

Mulock Houwer could in 1941 state (314) that after the introduction of sulfadrugs in Trachoma treatment there was a decline in the number of serious complications seen.

After World War II de Wyn used the frequency of Trachoma as an indicator of general health conditions, and compared the frequency in the interior (Toradja) of Celebes with that in coastal regions (1952, 589).

He found :

		Toradja			Coastal Indonesians	
boys, 6—15 yrs,	total	527	35.5%	Trach ;	323	50.5%
girls idem		280	43 %		188	57 %
all children		807	38 %		686	48 %
all older people		426	20 %		73	23 %

In the early fifties, after the discovery that antibiotics were active against Trachoma, it seemed for a time, that the eradication of the disease would only be a question of organizing mass campaigns, until it became apparent that also in this field the resistance against antibiotics emerged, and that the problem of reinfections remained if not all members of the family were treated at the same time. The next development was the introduction of vaccination against Trachoma : in this country Heath's thesis contributed to the knowledge in this field. In general the situation in relation to the possibility of vaccination against any disease, is stated as follow by the former Director of the Pasteur Institute at Bandung, Soemiatno (1963, 458). "If a certain naturally occurring infection does not leave significant immunity after healing, it is not to be hoped that a vaccine made against it will be of much value". (p.1) On vaccination against influenza: "The difficulty for us is that so many eggs are needed, which is a conditio sine qua non for production of the vaccine". The latter can maybe be obviated by the use of virus grown on cell cultures, but the former difficulty, lack of adequate immunity, remains both for Trachoma & influenza.

After 1963 progress was not as quick as could have been wished, and it can be gathered from the WHO's 1966 Report from the Scientific Group on Trachoma Research (567) that victory is not yet just around the corner, but that there are still many problems. I quote the Report :

"5.2. **The present status of field studies.**

A number of workers have reported that both live and inactivated TRIC agents protect human volunteers and simians against a challenge dose of the agent administered by the conjunctival route comparatively soon after the completion of vaccination. However, reports about the performance of trachoma vaccine under field conditions are conflicting. It is difficult to compare the findings of different groups of workers because of differences in the local epidemiology of trachoma and intercurrent eye infections ; in the age-groups selected for vaccination ; in methods of immunization ; and in systems for recording and assessing the results. The main conclusion that emerges from the various reports is that TRIC agents are comparatively poor antigens, and that a fully effective vaccine is not yet available. Although a measure of protection can be obtained against both experimental and naturally occurring Trachoma and there is some evidence of therapeutic effect, there is no assurance of solid immunity; several reports suggest moreover, that any beneficial effect is of a comparatively short duration. Nevertheless, very considerable progress is being made in fundamental knowledge of the PLT group, and there is hope that improved vaccines will result".

"5.4. **Publicity.**

There is a distinct possibility that unconfirmed and over-optimistic reports through public information media influence health authorities to relax prematurely their efforts to control trachoma by means other than vaccination. While recognizing that research on trachoma vaccine is a matter of great public interest, the group strongly recommends that workers in this field use their best endeavours to prevent the appearance of misleading publicity of this nature".

Trachoma in neighboring countries.

Figures on the relative importance of Trachoma in other Asian countries and in Australia can be seen from the WHO Survey on causes of blindness I quoted in chapter 15. There is some discrepancy between the frequency in surrounding countries and the fact that Trachoma in the Philippines seems to be rare (Fernando, 1934, 117). It is stated that among 1812 persons blind in one or in both eyes, only 0.5% was caused by Trachoma, whereas only 2 cases of Entropion in 14 years needed surgery. On Taiwan (Formosa) on the contrary Trachoma incidence was high (Fuchs. 132). After 1950 research on this island in cooperation with USA military facilities was intensive: in 1963 Woolridge and Grayston (1963, 581) published a report on trials with Trachoma vaccine among school children. Their conclusion : beside widespread vaccination also continued treatment with antibiotics of Trachoma cases is needed. The reason that neighboring groups of islands show such a difference in Trachoma incidence is not clear.

A very interesting method of investigating the different effects of environment on eye disease is used by Dhir c.s. (1967, 85) who carefully compared three eye diseases (Trachoma, Pterygium and Cataract) among the inhabitants of a village in Punjab, India, with the same diseases as they occur among racially pure Punjabi who have been living for decades in Canada and USA. For Trachoma it was clear that it was far less frequent and serious in Canada and USA than in India. Analysis of the data makes it probable that serious sequelae in Trachoma occur if there is much opportunity for re-infection, which is the case in heavily infected populations but far less in slightly or non-endemic areas.

Marré (1967, 272) uses another approach in that figures are compared for an area in Central Birma which was intensively explored, with figures from Leipzig Eye Hospital (Germany). In two districts in Birma, one with high Trachoma endemicity, the other with a low one, small children in the former area already showed a high Trachoma rate, whereas in the latter district the incidence rate with age was low to begin with, slowly rising with age. According to Marré, Bietti on Sardinia, and Winkler in Jordan saw similar occurrences. All of these data make it probable, that Trachoma is less dangerous as far as complications are concerned, which are after all the cause of visual impairment, in an area where the incidence rate is low, than in an area with a high Trachoma rate.

Basil Ward (1965, 546) investigated Trachoma on the Fiji Islands, with a view to judging the advisability of vaccination. He used the "social" classification of Ida Mann, i.e. Stage

A. Active Trachoma.
B. Trachoma healed with good eyesight
C. Trachoma with impaired eyesight
D. Blind from Trachoma

among other tools for judging the seriousness of the problem in Fiji.

The whole incidence of blindness was 1.19% of which ca. half was caused by Trachoma. The proportion of visual damage caused by Trachoma was naturally much higher, but rarely so high that it prevented the individual from working. For that reason Ward did not recommend a vaccination campaign, as the cost would be far higher than the useful effect to be hoped for.

Present importance of Trachoma in East Java.

The general impression among experienced oculists in this area is, that Trachoma is no longer what it formerly was: a real menace. One sees less and less of the dangerous complications, and fewer and fewer children with Trachoma. I compared the percentages of Trachoma cases seen at the Outpatients' dept. of Undaan Eye Hospital at Sourabaya in 1956 and 1966.

	1956	1966
Total number of outpatients	13.815	14.298
Trachoma in children under 16	14%	6%
Trachoma in adults	23%	11%

These figures concern Trachoma as a disease. From the Tables on Causes of **blindness** the following percentages can be calculated:

For children the number of cases of blindness by Trachoma is so small, that a "trend" is not discernible.

For adults in almost all groups the percentage is inclining, except for women in the oldest age group: Table p. 134.

In the varions tables I gave special a number to Trachoma and Syphilis combined (20. 1), because it seems that the combination is more resistant against treatment if antisyphilitic treatment is not instituted. From the older literature, which during the time of the investigation and even now, still has its value, I cite a paper by V. Derkac (1927, 82) who investigated the combination of Trachoma and syphilis and gives a number of other authorities who described such cases, many of whom saw suceess in Trachoma treatment only after having taken care of the syphilis factor also. From my figures it can be seen, that the combination does occur, but it is not very frequent in this area.

Comment.

At present there is no special effort in the direction of Trachoma control, except by the normal treatment with antibiotics locally, and applied by oculists, general physicians and nurses at dispensaries in smaller towns and villages. I am not aware of any mechanical treatment being carried out at present by any oculist. Besides, there is certainly a slow improvement in general hygienic ideas, dispersed by schools and other media. In various ways the general public has got hold of many kinds of antibiotic eye ointment, applied without advice, and of course there is a large amount of sulfadrugs prescribed for other ailments, but accidentally helping against trachoma at the same time, if it exists.

All of these factors and maybe more, cooperate in reducing the Trachoma rate and even the Trachoma-caused blindness. In view of the fact that mass Campaigns are costly in money and manpower, we can only be thankful for this unearned benefit. But it does not mean, that it can be the only solution of the problem, and that developments of new, easy and if possible, cheap methods of treatment and/or prevention must not be followed carefully, until there emerges a method which is suitable and applicable in this country. Meanwhile all sorts of hygienic measures are to be applied as carefully as possible, e.g. water supply, and the best available treatment is to be carried out by as many kinds of medical and paramedical personnel as can be trained.

	1958	%	1959	%	1960	%	1961	%	1962	%	1966/7	%	
Total Trach.	284 31	11%	246 24	10%	214 23	11%	205 30	18%	190 14	7%	106 5	5%	Male, 16 — 41 yr.
Total Trach.	232 34	15%	231 38	16%	223 42	19%	218 40	18%	187 28	15%	111 8	7%	Female, 16-41 yr
Total Trach.	278 44	25%	159 38	24%	189 51	27%	204 46	22%	212 35	16%	120 14	12%	Male, over 40 yr.
Total Trach.	160 47	29%	198 50	25%	214 58	27%	208 61	29%	211 54	25%	111 26	23%	Female, over 40 yr.

Chapter Nineteen.

Retinoblastoma.

Retinoblastoma deserves a special chapter, not because it is important as a blinding disease, but because it is a killing disease as well.

In Indonesia it is not rare : in prewar times there were several case presentations, and it is mentioned in a paper of Dr. B. Bakker on eye tumours and in a paper on retinoblastoma alone by Mulock Houwer. Bakker (1932) collected 5 cases which had been seen in 5 years at Bandung Eye Hospital among 34.851 patients (0.015%). Mulock Houwer saw at Batavia an incidence of 0.14% among the patients of the Eye Departement (312). Surbek (1932,492) demonstrates one case at Palembang, F.C. van der Horst (1938, 176) had several cases at Malang, twice with tumours in both eyes. Wempe at Malang (1940, 555) showed a patient who after enucleation for glioma retinae died of a carcinoma of the parotis. Both tumours were examined by the pathologist. Oey Khoen Lian saw in 1941, (331) at Batavia a case which died of distant metastases, i.a. in the femur.

After the War Maäs at Medan (249) saw even 7 cases in something over 2 months in 1959. These cases are described by him and the pathologist Kern (199).

Usually the tumour is situated in one eye only, but a certain number is binocular: according to Mulock Houwer (1941, 312) at Batavia even in 40% of cases.

It is unusual that there are metastases, mostly the tumour grows per continuitatem : Oey's case was an exception.

According to Duke-Elder the white race has a higher incidence of retinoblastoma than pigmented races, but several cases have been described in dark races. Sherman (430) discribes one in an American Negro child, which presented as a phthisis bulbi. Khosla e.s. (202) mentions an Indian child, where the picture of a panophthalmitis was simulated. Kodilinye discribes a high incidence at Ibadan, Nigeria, 1967 (209) of 0.52%.

Usually the first signs of the tumour are a white or yellowish mass visible behind the pupil, but cases of : uveitis, hyphema, hypopyon, phthisis bulbi and panopthalmitis of unknown origin should be suspect if they occur at a young age. If the tumour has already progressed to the size of the child's fist, as is not rare in these regions the diagnosis is not difficult any more.

We had difficulties in one case, where I suspected a retinoblastoma, possibly of the infiltrative type as described by Manschot, 1955 (269) and Schofield, 1958 (418). The latter performed aspiration of the aqueous for examination for tumour cells. In my case the result was negative. After enucleation the pathologist Moeljono found dictyoma. In a few years time several cases of dictyoma have been seen at Surabaja. This tumour also can be dangerous, as it grows into the cranium, although it looks benign.

In Indonesia the real incidence of the tumour cannot be ascertained, as it is quite sure that many cases never reach an eyedoctor.

I know of 2 families where retinoblastoma was discovered, and where the parents afterwards said, that they had already lost a few children by the same cause : in one family it appeared that 5 of 8 children suffered from this disease, in another 2 of 5. Nevertheless the parents came in a very late stage with their most recent child with retinoblastoma. In these cases consanguinity was not admitted.

In the Netherlands, where Mrs. Schappert and Dr. Hemmes (415) have published data on almost all of the cases of the last 100 years, i.e. 550 cases, it can be stated that the real incidence is 1 case on 15.000 births. It is not at all certain that the incidence would be as high here, because in the Netherlands there are already children and grandchildren of cases who have been successfully operated upon. The incidence among offspring of retinoblastoma cases is unfortunately rather higher than usual, especially the tendency for binocular cases increases. In Indonesia retinoblastoma can still be considered a lethal factor : the number of children who come in time to save their lives is still very small.

As a cause of blindness Retinoblastoma figured in our present study as follows : 1958 — 1962 : boys under 17 yrs. 5% ; girls 1.7%
1966 — 1967 1.6%

This compares to : Schappert in the Netherlands, survey, 1959 : 6.2% USA : Kerby (cited by Schappert 414) 2.4% Hatfield (169) 1.7%.

Among cases of **monocular blindness:** Undaan 1966/7 : boys 6.4%, girls 7.8%. This means that in one year's time there were seen : 13 cases of Retinoblastoma among ca. 13.000 outpatients. Many of them were not referred by physicians.

Therapy. Classical therapy is still enucleation, but with various kinds of irradiation good results can be obtained, even making it possible to save the eye, which especially in binocular cases is very desirable. **Prevention.** Essentially prevention is not possible. Most cases are sporadic non-familial cases, theoretically only in familial and hereditary cases something could be done by eugenic measures.

In this country, where the knowledge of the possibility of this fatal disease is seemingly not widespread, it would be very useful if all types of medical and paramedical personnel during their training and if possible afterwards, were warned of the possibility of recognizing it : every case in a small child where something seems to be wrong behind the pupil, should be sent to the nearest eye doctor as quickly as possible. Even if a few cases of congeniatal cataract or ordinary uveitis are referred, that would not be unuseful also, on the contrary.

Chapter 20. Cataract.

Cataract as a cause of blindness can give rise to astonishment : usually persons who are blind by senile cataract are only temporarily so, as they can be helped by surgery, often with complete restoration of eyesight.

This is the reason, that statistics on blindness caused by cataract can vary widely, according to whether ALL cataracts have been counted, or only those, where surgery was refused, or unsuccessful, or where there was a contra-indication against surgery. Much depends also on the country where the statistics are collected : in a country like the Netherlands, with many towns with competent oculists, which is so small that one is practically never farther than 3 hours' easy travelling from an oculist, where public opinion is agreed on the idea that cataracts must be removed and where, last but not least, social legislation is so far odvanced, that financial conciderations never enter into the problem of medical help any more, the figure for cataract as a blinding disease will naturally be low, according to Mrs. Schappert-Kimmyser (1959, p. 53. 414) around 5% of total blindness. In Indonesia on the contrary, where there are many large and small islands, among them even large ones, where there is only one oculist or none at all, where travel has various kinds of difficulties, and where large sections of the population are very longsuffering and do not tend to seek help quickly, a relatively small proportion of the existing cataract cases will come in the hands of an oculist. Our patients at Surabaja often have travelled for a day or more by land, or even for weeks by sea before reaching the nearest eyedoctor. Small wonder that they will think for a long time before undertaking the arduous journey, which is all the more arduous because of their blindness. There is no financial aid to be expected for sick people except from their relatives, who often need a long time to collect sufficient cash : although the economy is changing from barter to a money economy, this does not mean that the average villager can easily lay his hands on ready cash in any amount: transport is the problem as it is expensive, which is not balanced by the hospital and the eyedoctor asking only a nominal fee.

From our hospital records it is thus difficult to get an impression, not of the number of cataracts we see, but of the real frequency of cataract. Before the last War the general opinion among oculists was, that cataract here was not more frequent than e.g. in N.W. European countries (Tyssen 524, Bakker 1932, 18, Mingelen 284). The shape of the usual cataract here and that encountered in Europe is different, as described by Mingelen in his thesis (284). The cuneiform lens opacities which are frequent in Europe are rare here, whereas here the opacities are often nuclear, and still more frequently situated centrally in the posterior cortex. Mingelen compares that latter form of cataract to "glassblowers' cataract" and mentions tropical light which supposedly has many infrared components as the possible cause of the difference.

The figures which were found by van Manen (1938, 262) and Gomperts (1940, 151) both on Sumatra, were neither high nor low. A factor which enters into the problem is the life expectancy of the population, as Schubert (1964, 420) who worked on New Guinea (now W. Irian) has pointed out. He, and also Ida Mann (267.) in E. New Guinea found very low figures for cataract, probably because the population does not reach the age where cataract flourishes. It is rather difficult to estimate age in Indonesians : Those who have been more or less well-to-do from birth, and who probably have been properly fed, can look astonishingly young for their age, whereas the agricultureal laborers look too old for their age as stated. Müller (294) tried to apply part of the signs for estimating age as used in the system of Nadeshdin to older Javanese people from a village which had been christianized long ago, and where consequently the people were registered at birth : in this group of persons he found that the age estimated by the appearance of certain skin folds and wrinkles of face and hands was lower than the real age of the persons. Müller ascribes this phenomenon to an anatomical difference in the skin layers of pigmented skin.

Hard work and inappropriate food seem possibly to be the cause of early aging in developing countries. In India, where in certain regions cataract must be very frequent, also tropical light is accused as an additional factor. In Indonesia conditions are not the same as in India: there have been investigations into the amount of sunlight, which is high all the year through compared to countries farther removed from the equator (Boerema, and Berlage. 39). Indonesia is essentially in most of its islands a green country, and therefore there is little "glare" as is described from India by Elliot (104). The composition of the sunlight in its various wavelengths is not measured continuously, but Vry (540) has measured the amount of ultraviolet rays at different heights on Java, and found that even in the coastal plains there is more ultraviolet than in moderate climates. On the mountains when it is clear weather the amount is naturally even higher. In the plains during part of the year there is a kind of turbidity of the air, caused by moisture and dust in the atmosphere especially in the second half of the dry monsoon, and it is probable that ultraviolet rays are hindered from reaching the earth by this "blanket". Whether there exists a difference in frequency of cataract between the plains and the mountains is not yet known. It would be very useful for an investigation into the causes of cataract (and possibly also of Xerophthalmia) if research into the spectral composition of the sunlight as it reaches the earth in various parts of Indonesia could be instituted. Possibly this would be of use also for agricultural purposes.

Before the war already, and generally in other tropical countries also, the general opinion is that various eye conditions, which increase with age, have an earlier age of beginning than is usually seen in temperate zones. Cataract, Presbyopia and also inversed astigmatism come under this heading. Changes in general hygiene and nutrition seem to have an influence: Kajiura (1965, 194.) reports from Japan, that Presbyopia which in 1919 began at age 43 now begins at age 47. From this fact it seems we can conclude that the colour of the iris is not of importance in this context, as Japanese before and after have deepbrown eyes. Rambo and Sangal (1960. 388) constructed a curve for the loss of accommodation: this curve starts at the same age in Indians and Europeans (Duane's standard), but it shows a gradual loss of power of accommodation in white people, and a sudden loss in Indians, resulting in the fact that the latter need reading glasses 1 dptr. at 40, whereas generally this strength is needed in Europeans at age 45. These figures agree well with those found in Indonesia (Westhoff 563 de Ruyter 405).

In the Panama Canal Zone Covell (66) saw that (white) USA labourers and (black) Panamian workers, who lived under the same conditions, also got the same food, showed a difference in age of onset of Pressbyopia : Panamians needed reading glasses 5—10 years earlier than Americans, and this need arose abruptly, so that they need 2.5 dptr. at an early age. According to Covell the only difference between the two groups was, that diseases like tuberculosis and syphilis were more frequent among the Panamians. To which one could add : probably the Panamian group had lived always under tropical light, whereas the (Northern) American group was exposed to it only later in life.

It can be expected that there exists a certain parallelism between the age of onset of cataract and that of presbyopia, as possibly the latter is related to increasing hardness of the lens with aging (and of the ciliary body?). The real cause of both conditions is not yet clear, one can only collect data on the influence of possible factors which differ in populations where they begin early and populations where they start later, and hope that one of these factors, or probably a combination of them, will finally emerge as the true cause.

Marré in Burma (272) saw that at age 50 already 13.5% of cataract cases in Burmese had vision of 5/50 or less, whereas at Leipzig for age 50 the percentage was 1.4. Dhir c.s. (85) who compared Punjabis in their native surroundings with Punjabis who had lived in Canada and USA for decades but where racially pure, saw that cataract in India appears at an earlier age and has a higher prevalence, and that there existed a correlation with solar radiation. It was striking that people who had lived for 40 years or more in India and who finally migrated to the American continent, showed the same high prevalence of cataract as if they had stayed on in India. Probably the influence which causes cataract is not a short time influence, but an accumulative one, if active over a long period.

Cataract in neighboring countries.

The figures which are to be found in the WHO survey on Causes of blindness (568) and which I quoted before, are to be read with the reservation mentioned above, i.e. that it is mostly not stated if all cataracts are counted, or only the finally blinded cases. In all of the neighboring countries cataract is to be found, but comparison of percentages is not feasible.

Cataract in Indonesia.

Mingelen (1935, 284) gives an analysis of the problem of cataract on Java at the time in his thesis. Besides, two related problems seem to have been discussed in the thirties : as well Mulock Houwer (296 a) as Wille (575) give a warning for the danger of glaucoma developing in mature cataracts, and recommend therefore operation of every mature cataract, also monocular ones. Generally one is reluctant to operate on monocular cataract, if eyesight in the other eye is still good, as mostly the patient is disappointed with the result. In circumstances as they prevail in this country, then and now, one has to take into account, that the patient will probably not be able to come for regular check-ups to take the intraocular tension, because of the distance between his home and the doctor, the costs involved in travelling, and the fact, that people are often unobservant and cannot be made to realize the risk they run. It is sound practice here to operate on monocular mature cataract if there is no garantee that the patient is able and willing to come for regular check-ups.

The other, related, question which was discussed in the thirties, was if it was to be recommended, that general practitioners in faraway regions would undertake cataract surgery, to relieve the patients from travelling, and from needless suffering if they could not be brought to a centre. Tyssen (524) who as a travelling eyedoctor operated on hundreds of cataracts under "village conditions" said that his work was welcomed everywhere, and some general practitioners (Bloemsma 32) found they had success also if operating in small towns. Gomperts (151) compared the results of Tyssen's work in Atjeh with that of eye surgeons who worked in a decently equipped centre, and found the results of the latter much better. From the discussion emerges the conclusion, that the first condition which must be met in cataract surgery is pre-operative examination and post-operative treatment which should be good. They make or break the work of the surgeon, (van Manen 262 Maäs 247 Mulock Houwer, Oey Khoen Lian 332).

Cataract in East Java.

From the tables of classified causes of blindness in the present study, one can conclude that cataract is a moderately important eye disease in this region. In the Tables 1958—62 I included not all cases of cataract which came to the hospital's outpatients' department, but only those which refused operation, where there existed a contraindication against surgery, or where surgery had been unsuccessful ; unfortunately also a few cases had to be included which had been operated upon by unauthorized and unqualified "help" outside. To get an impression of the true number of cataract cases, I counted them in the year 1960 only, using the same limit of visual acuity as usual, i.e. less than $1^1/_2/60$ in the better eye. I found 450 cases, in 14 of them diabetes was found also (routine examination of all cataract cases). They were for the largest part operated upon successfully : the others are entered in the lists for 1960.

The figures for the years 1958—62 were finally established after a few years had passed, but for the one year-period 1/8'66—1/8'67 this could not yet be done, as the time for folllow-up was still too short. That is probably one of the reasons the percentage for cataract in this latest period is higher than that in the former : it is to be expected that a number of cataract cases are still "thinking over" our proposal for surgery and will eventually turn up to be operated. I made two lists, one including all cataracts as seen in the latest one year-period, and another only including cases according to the criteria as applied in 1958-62, i.e. listing only the cases which were unsuccessfully operated upon, those with a contra-indication, and the patients who refused surgery. As the follow-up is not yet complete there is no sense in computing "trends" in the percentages of cataract in the earlier and in the later period.

Congenital cataract.

The situation in congenital cataract is somewhat different from that in senile cataract. We saw a definite increase in cases of congenital cataract during 1959 and 1960, part of them showing congenital heart disease also. One would be tempted to think that there had been a Rubbella epidemic previously, as we know since 1941, that congenital cataract can be the result of Rubella of the mother early in pregnancy (Gregg, 1941, 147). Unfortunately Rubella in this country is a diagnosis with special difficulties : the rash often is present only for a short time and not easily seen in a brown skin, and other signs and symptoms are not alarming enough to bring the patient to the doctor. Only a very few mothers of our patients with congenital cataract seemed to remember that they had been suffering from a fever early in pregnancy, but many more could easily have forgotten such an episode, as their indisposition would have occurred one to one and a half year previously to the time they brought their children for examination and were asked after it. Beside Rubella another possible cause could have been the so-called Asian 'flu, but the time of that epidemic did mostly not fit into the picture. Rubella, according to the pediatricians, is sometimes found in this country, but it is not frequent, and general practitioners will possibly have difficulty in diagnosing it : the symptoms which clinch the diagnosis are enlarged occipital glands and plasmacells in the bloodsmear, the latter only present for a short time. Recently it has been possible to isolate the virus (Parkman, c.s. 1962, 354), to grow it in tissue culture (Weller, c.s. 1962, 554) and to develop serolocgial tests for it (Alford 1964, 7). The virus has even been recovered from the cataractous lens material at operation (Roy c.s. 403. 1966, Cotlier c.s. '66, 64) which is successful in rather a high percentage of cases (Murphy c.s. '67, 316). It is as yet not possible to perform these tests in this country ; as soon as they can be done, the diagnosis cataract caused by maternal Rubella will possibly have to be included in our lists.

Hereditary cataract.

Congenital cataract with a hereditary origin is not exactly frequent here, but we found it several times. It can be a blinding disease, if it is combined with other ocular malformations as is not rarely the case, e.g. microphthalmus. Go (149) described a family with this hereditary trait and isolated cases of the Marfan syndrome with cataract we saw twice in the 1958- 1962 period, which normally is a hereditary disease also.Fast (110/112) and van der Horst (175) have described families, indicating that it is found all over Java.

Chapter Twenty one.

Glaucoma.

Glaucoma in this country is one the most treacherous eye diseases, under-estimated as well by medical personnel as by the general public. Except for the congenital and juvenile forms, which are not frequent, generally Glaucoma is a disease of middle and old age, and is therefore dependent for its prevalence on the life expectancy in a country, and on the the onset of aging. In countries where life expectancy is high, the frequency of Glaucoma is also high, e.g. in Icelend (568), where another factor comes into play, i.e. heredity, because of frequent consanguinity.

In Indonesia generally life expectancy is not high : Su'dan (1967, 488) states for Makasar 42.3 years at birth: aging seems to start earlier than in moderate zones, if we may judge from the frequency with age of such affections as: Presbyopia (Westhoff, 1911,563 ; de Ruyter, 1919,405;) Cataract (Mingelen 1935,284); inverse astigmatism which according to an investigation of my own shows an increased frequency at a much earlier age than is usual in Europe (Kongres Perdami, 1968). As far as I am aware careful research into the incidence with age of gerontoxon and pinguecula has not yet been instituted in this country.

Whereas Westhoff (1911, 563) and Louwerier (1924, 245) already stated that Glaucoma was not rare, and Bakker (1932,18) found that acute Glaucoma was as frequent here as elsewhere, Mulock Houwer (1940, 311) said it was rather rare, in distinction from frequent secondary Glaucoma. Sie Boen Lian ("Tropical Ophthalmology in Indonesia and adjacent Countries", Adv. in Ophth. vol. 10,1960) and Nana Wyana Djaja Koesoemo ("Research in Indonesia, 1945-1965") copy this, in their otherwise valuable compilations. The latter adds that refined modern methods of diagnosis seem to contribute to a rising frequency of Glaucoma, but as Elliot already pointed out in in his book (1922,194) the Glaucoma cases usually seen are so obvious that refined methods are not (yet?) needed for their diagnosis.

At the present time it certainly is not true any more that primary Glaucoma is rare, and in our experience of the last ten years it is more frequent than secondary Glaucoma. The classified lists of causes of blindness show :

	male	female
16-41 yrs age group	3.6%	10.2%
over 40 yrs age group	16.6%	30 %

All of these concern primary Glaucoma, in some cases only combined with cataract where it was not possible to find out which was the cause, which the effect. No cases of Glaucoma secondary to leucoma adhaerens or uveitis are listed under Glaucoma.

What would possibly be the cause of the difference in frequency as seen in a large eye hospital 30 years ago and now? In East Java at least life expectancy certainly has risen somewhat, especially after the malaria eradication compaign, and possibly also by somewhat more effective treatment of infectious diseases, by the advent of sulfa-drugs and antibiotics. On the other hand there are at least two reasons which induce people to overcome their reluctance to seek medical help sooner than formerly: economic pressure in the villages does not allow any more that people are sick and improductive, which is helped by a greater trust in the possibilities of modern medical treatment. The latter is an important byeproduct of the Yaws eradication campaign, which has impressed the people by its quick effectiveness. The combination of all of these reasons which were not operative in prewar times, may account for the different situation in the frequency of glaucoma as seen in the larger eye hospitals at present.

It is a very unfavorable factor, that glaucoma as a separate disease is not wellknown, whereas cataract is very wellknown. Because of the similarity of both diseases (slow loss of vision in older people) they tend to be mixed up, as Bargy, (1929,20) already saw in Indo-China also. The idea that cataract will not be operated before the person cannot see at all is very widespread. Whoever has glaucoma (or optic atrophy for that matter) must wait also until he cannot see at all, at the advice of neigbors, village elders, possibly even of midwives and nurses in charge of dispensaries. In that way the time for effective help will have passed before the patient reaches a doctor, but because of the expensive yourney the advice will be gratefully followed. In the education and training of every kind of medical personnel from doctors to midwives it should be stressed, that it is necessary to have an expert opinion on all cases of slowly deteriorating vision in older people, and not to postpone it, as treatment of glaucoma can only be effective if started early ; ohterwise the loss of visual field will be so large as to be disastrous.

Frequency of glaucoma.

In most countries glaucoma is a rather frequent eye disease. Exceptions seem to be New Guinea (Schubert 1964,420 Ida Mann & J. Löschodorfer, 1955,267) and Australian aborigines (Ida Mann, 1954, 265/6). Lépine (1957,232) states that at Phnom Penh glaucoma is rare also, although it seems improbable in the surroundings.

In countries in moderate zones it is generally found that the incidence of unsuspected glaucoma is ca. 2% in the population over 40 years of age. Leydhecker (1958,233) examined 10.000 persons of all ages, but most of them between 30 and 60 years of age, who had no eye complaints, by tonometer. The incidence of glaucoma diagnosed by this and other methods was :

age	20—29	30—39	40—49	50—59	60—69		Over 40 yrs
male	0.43%	0. 8%	1.74%	3.17%	5.33%		2,72%
female	0. 2%	0. 4%	0.75%	1.75%	—		1.07%
All	0.35%	0.65%	1.45%	2.84%	4.48%		2.31%

Frydman (1966, 128) reports a glaucoma survey among the population of Florida (USA) concerning 67,000 persons, where 1.4% had glaucoma, of whom 55% were younger than 55 years, 63% between 55 and 75 years, and 17% over 75 years.

In Israel Blumenthal & Kornblueth (1965,35) found that of 4363 seemingly healthy eyes 1.73% was glaucomatous, 1.6% in men, 1.9% in women. If Jews originating from Oriental countries were taken separately their glaucoma percentage was even 4.1%.

Molnár et al. (1967, 289) examined the whole population of the community of Ujléta (Hungary) and found glaucoma in all ages 1.28% of which 37.1% were men and 62.9% women.

The firs oculists in Liberia came from Israel for a few years. They found in a survey among the population, concerning 1426 persons, 3.25% glaucoma (Neumann et al 320).

In outpatients' departments naturally the percentages found for glaucoma are higher than population surveys. In Liberia among 12.000 outpatients in those over 40 years of age 14% had glaucoma, among the blind 24% was caused by glaucoma. This was never angle closure glaucoma, it often began under 40 years of age, and high hypermetropia was frequent.

In other Negro populations the impression often is that glaucoma is frequent also (Degazon, Jamaica 1952, 79) but according to questionnaires distributed by Mc. Nair among USA oculists opinions were divided. Sarkies (Goldcoast, 1956, 410) found the same frequency for primary glaucoma as is found elsewhere.

Glaucoma seems to be frequent too in other pigmented races: Elliot (India, 1922, 105) Meyerhof and Peretz (Egypt, 1928, 282.361) MacCallan (Egypt, 1922, 252) Bargy (Indo-China, 1929, 20) Fernando (Philippines, 1926 116) Bartels (Turkey, 1918, 21), Pillat (China, 1932 370) Chang Hsiao-Lou (China, 1951, 52). Kagoshima (1916. 193) only found in Japan frequencies which were more of less the same as those found by Haag in Germany at the time, according to Elliot.

All of these authors, except for Kagoshima, point out, that glaucoma in their regions begins at an earlier age than is usual in Europe.

For the years 1966 and 1967 I collected all cases of primary glaucoma seen at the outpatients' department of Undaan, Surabaja, and found the following frequencies with age (those under 20 years excluded) :

	20—29	30—39	40—49	50—59	60—69	70—79	80 over	Total
men women	9 15	23 44	37 88	43 81	33 32	3 12	5 1	153 293
All	24	67	125	124	65	15	6	446

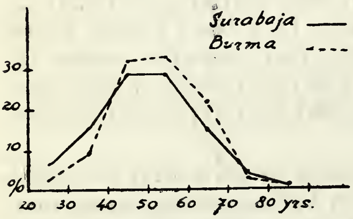
In percentages by decades :

men	5.8%	15 %	24.1%	28.1%	21.5%	1.9%	3,2%	34.4% of total
women	5.1%	15 %	30 %	27.6%	10.9%	4 %	0.3%	65.6% of total
All	5.5%	15.3%	28.5%	28.5%	14.9%	3.4%	1.3%	

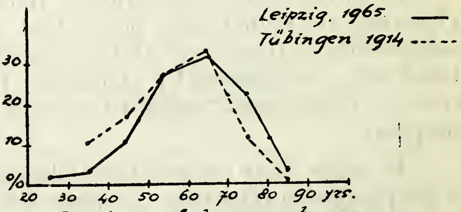
For Burma, Marré (1967, 272) gives the following percentages by decade:
2% 8.6% 31.2% 32.3% 21.4% 3.2% 1% total 904 patients.

and compares them with Leipzig in the same period of investigation:
1.2% 2.6% 11.8% 28.4% 31.7% 21.4% 2.8% total 1075 patients.

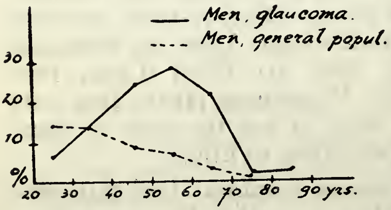
C. Haag (1915, cited by Elliot 155) found in 1032 consecutive cases of of glaucoma at Tübingen (Germany) the following percentages by decade :
11.6% 17% 28% 32% 11% 1% total 1032 patients.



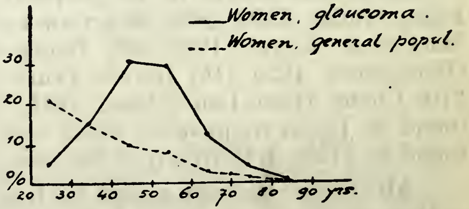
Incidence of glaucoma by age, Surabaja, 1966-1967, Burma 1965



Incidence of glaucoma by age, Leipzig - 1965. Tübingen 1914.–



Incidence of glaucoma by age, men, compared to composition of general population, men.–



Incidence of glaucoma by age, women, Compared to composition of general population, women.–

Fig. H

In fig H these percentages are shown in a curve. It is striking that the curve for Germany is so stable over 50 years: only the percentage for the highest age group is higher now than it was before the First World War, possibly because there are more old people now. The curves which represent the frequency of glaucoma with age in Burma and Surabaja are almost parallel also, but the rise in frequency lies a full decade earlier than in Germany. (statistically significant by the X^2 method). As the percentages in Japan at that time were similar to those in Germany, it seems improbable that the eye color as such is a causative factor.

Weinstein (1953, 553) mentions that between the ages of 55 and 64 years, the narrow angle type of glaucoma is more frequent, whereas in the decade 65-74 years, especially in men, wide angle glaucoma prevails. It seems possible that this contributes to the female preponderance in glaucoma in connection with the low life expectancy, which usually is somewhat higher in women : there are enough women, who get old enough to suffer from their type of glaucoma, i.e. the narrow angle glaucoma, but not enough men become old enough to get their special type of glaucoma, the wide angle glaucoma, which emerges only at a higher age, which generally is not reached here.

The frequency of the various types of glaucoma vary somewhat. Weinstein (1953, 553) gives statistics over 30 years, Budapest, comprising 3,214 patients.

	inflamm. ac.	inflamm. chron.	simplex
Weinstein	18%	72%	10%
Sugar (889 cases)	14.8%		
Sugar (299 cases in other hospital!! 1958)	6%		
Lehrfeld & Reber (1937, cited by Sugar 489)	22.7%		

Lehrfeld & Reber give the following percentages per decade for angle closure glaucoma: 40—49: 11.5%; 50—59: 31.1%; 60—69: 36.1%; 70—79: 18.7%: over 80— 2.5%. If we apply the same argument as for Indonesia above, it is possible to suppose, that the lower figures of Sugar are caused by the higher life expectancy in USA which developed between the time of the investigation of Lehrfeld and Reber and that of Sugar.

Dalgleish and Naylor (1965, 71) give percentages for age for surgery cases of glaucoma, which are in general agreement with the percentages found in Europe for narrow angle and wide angle glaucoma.

Molnár in the investigation of the whole population of a Hungarian village found that of all glaucoma cases 37% were men, 63% women. This does not agree at all with the figures of Leydhecker, who found twice as many men in his survey among seemingly healthy persons. Our figures for Surabaja (34.3% men, 65.7% women) are in close agreement with those found by Molnár and formerly by Haag and Kagoshima.

In fig. H our percentages of glaucoma per decade are compared to those for the population at large. The earlier beginning of glaucoma in women, which in the 5th decade already causes 30% of all cases, compared to 25.8% in men, is statistically significant by the X^2 method, for 0.5 level.

Weinstein further mentions, that of narrow angle glaucoma 20% was seen in men, 80% in women, and that generally narrow angle glaucoma is related to essential hypertension, wide angle glaucoma to arterisclerosis.

Diagnosis of Glaucoma.

To be able to diagnose pathological differences one should know what is normal. Ocular tension: Leydhecker found for the normal ocular tension a binomial curve, from 7.5-26.4 mm Hg, mean value 15.5 mm Hg. With age this value is very slightly rising, less than the standard error of the tonometric method. Rigidity of the sclera also slightly increases with age, only significant between the 2nd and 7th decade respectively.

Taubenhaus (1967,502) examined 5,687 eyes of glaucoma-free persons between 40 and 85 years and over, and saw no correlation between ocular tension and age, nor in the difference of tension in both eyes. Mansours F. Amaly (1965,270) in the USA on the other hand, found an increase in ocular tension with age, especially in women, which was statistically significant: in men the tension rose on an average from 14.93 mm Hg to 16.14 mm, in women from 14.97 mm to 17.15 mm Hg. In persons who had a relative suffering from glaucoma the average pressure was also higher. Davanger and Holter (1965,75) in Norway found for the average ocular tension among the population not a Gauss curve, but a skewed curve of dispersion. This could be caused by subclinical cases of Glaucoma being mixed under the healthy eyes, but they compute mathematically, that it is possible also that variations in normal eyes causes the skewedness. Kitahata (1916, 206) found that the normal ocular tension in Japanese in 1,0 mm Hg lower than in Europeans. Eisa Hamdy (Egypt, 1939, 102) states that the normal intraocular pressure in Egyptians is the same as that in Europeans.

The sometimes seen rise in intraocular tension on strong emotions, as e.g. in Jews at the time of the progroms in prewar Germany, is possibly connected with a sudden rise in blood pressure.

Visual field. In races with a lowbridged nose, the visual field is larger in the naso-inferior direction than is seen in the standard charts for European eyes (Fernando, Philippines, 1926,116; Chung et al. China, 1965,56).

Pupil size: Obianwu et al. (1965,327) found that the pupil size in dark eyes is smaller than in blue and grey eyes: Trendelenburg, (cited by Bürger, 1958, Acta XVIII Congr. Ophth. Belg. p. 280 seq.) states that the pupil as well by day as by night steadily decreases in size with age.

For the diagnosis of glaucoma are important some early signs, among which slowly developing nightblindness, shallow anterior chamber, early loss of accommodation. Except in population surveys, which usually depend on the tonometer, confirmed afterwards by other methods of examination, the diagnosis of glaucoma usually is based on: specific complaints, redness of the eye, haziness of the cornea, large, oval, inactive pupil, high ocular tension, cupping of the disc. In the judgement on all of these characteristics the values and conditions which are normal in the region should be taken into account. More detailed descriptions of modern investigative methods in glaucouma diagnosis can be found in the paper by Prof. Salim (1962, 406).

Glaucoma as a blinding disease.

Magnus (1883 231) found in Germany among all age groups among the blind 8.97% glaucoma, Hübner (1922 231) 4.75%. Dickson (1946,86) in Scotland found 8% among people certified as blind, Alaerts (1952,6) found 20.7% among the blind of Brussels, Belgium, Schappert-Kimmyser (1959,414) 9% among adult blind in the Netherlands, Vedel-Jensen (1962, 531) 15.2% in 1000 consecutive new members of the Danish Society for the Blind, Kasparow (1965, cited by Molnár 289.) 45.5% in Archangelsk, Russia, and Sorsby (1966,472) 12.6% among the registered blind of England & Wales (14.5% in men, 11.5% in women). On other continents with Caucasian inhabitants were found: Fitzgerald (1949, Illinois USA 123) 14.2%, Meek (1950, New York State, USA 280) 17.3% (blindness was a reportable disease during this investigation). Bruce Hamilton (1951, Tasmania. 165) 14.4%, Yates (1963, Australia 595) found more or less the same causes of blindness and the same frequency as Sorsby in England.

Other figures can be found in chapter. fifteen.

Generally glaucoma is not rare in developing countries, and as the people tend to come late for help, it is important as a cause of blindness. MacCallan (1922, Egypt 252) saw among his glaucoma patients at first examination 75% who were already blind in one or both eyes. Pillat (1932, China. 370) found that 31.2% of his glaucoma cases were already blind in one eye, and even 48,5% in both eyes. The general experience in Indonesia is the same.

Therapy of glaucoma.

Glaucoma can be treated with miotics in an effort to lower the introcular tension, and if necessary surgery has to be applied. The general rules for treatment of glaucoma are the same everywhere. But in view of the fact that the disease regularly will strike in both eyes, even if the time interval can be years, and in view of the fact that people tend to come too late, it is wise to operate on both eyes also if only one shows signs of glaucoma. This practice was recommended already by Elliot, also by Louwerier (1923, 245) in Indonesia. I prefer a peripheral iridectomy with cautery on the seemingly healthy eye, which is simple to perform and innocuous. It can happen that the visual acuity of the socalled healthy eye will even be improved by this procedure, which, of course means that the eye was already glaucomatous.

Ronas (401) and Somerset (1963, 466) state that dark eyes often will stand a high tension surprisingly long, but there are many surgeons who are of opinion, that especially the Negro eye stands fistulizing operations not well at all (McNair 1951,258; Abraham, 1958,2; Sédan, 1964,423; Hall, 164; da Silva, 446). Sometimes the higher frequency of Syphilis in the Negro is held responsible, but mostly the wellknown tendency of pigmented races to keloid formation in various kinds of cicatrization is thought to be the cause of early closure of the trephine opening. Unfortunately it is difficult to compare results of surgery here with those achieved in moderate zones, as the degree of Glaucoma at the time of surgery is very different.

Except in cases who live near and who can be trusted to come regularly for check-ups, it is a general rule that one cannot rely on miotics, but should have resource to surgery immediately. This is not a medical indication in some cases, but a socio-economic one, which has already been pointed out by Elliot, and again by Sarkies (1956,410) for the Gold Coast.

Factors which are supposed to be instrumental in the high prevalence of Glaucoma in developing countries:

Color of the iris. There is an old belief, that brown eyes more often are suffering from Glaucoma than do blue or grey ones (Tyrrel, 1840, Sichel, 1841, Rydel, 1867, all of them cited by Schmidt-Rimpler, 417). But Gama Pinto found the same frequency of Glaucoma in brown-eyed Portuguese as was found in blue eyed peoples (1906,374). McNair brings the point up, but otherwise it is seldom mentioned in the recent literature.

Anatomical dfiferences. Cunningham (1948,69) states that the iris of the Chinese differs from the European one in that the anterior limiting layer is tougher, the whole iris structure less spongy, and the iris root thicker than in Europeans.

Physiological and pharmacological dfiferences. Brown eyes generally have a smaller pupil in rest than blue eyes, and they react slower to mydriatics. Obianwu et al. (1965,327) compared the effect of epinephrine in the eyes of European, Indian, Chinese and African students and found that the mydriatic effect was inversely proportional to the degree of pigmentation of the iris.

Heat. Lobo (1914, cited by Peters, 363) felt that Glaucoma was oftener seen in hot countries, because the heat favors cataract formation, which in its turn can lead to Glaucoma. Kasparow (1965,196) in a population survey in Archangelsk, Russia, found more Glaucoma in workers in hot surroundings, as was confirmed by Molnár et al. from Hungary 1967,289). In moderate zones on the other hand acute attacks are more often seen in winter (Weinstein, 1953,553). Elliot (105) and Kagosjima (193) said the same. Maynard at Calcutta saw more cases in the rainy season (1908,279) attributing this to the sudden change from strong light to a darker sky. It would seem that it also could be explained by cold, wetting, meteorological factors. Sautter (412) found a accumulation of Glaucoma attacks in certain types of weather, involving front passages and turbulence, with upglide movement of the air. According to Mulock Houwer in Amsterdam at the same time as an accumulation of Glaucoma attacks occurred there were frequent cases of eclampsy. The weather type is in Alpine countries known as Föhn weather, and is nearly the same as that which occurs on Java in the period of change between the dry and the wet monsoon, just before a thunderstorm when the heat and humidity can be highly oppressive ("Kentering"). Van der Schaar (1940 GTvNI, p. 706 seq.) studied many aspects of meteorology in relation to certain types of diseases.

Nutrition. That there exists a connection between the early aging in the tropics and the often encoutered state of chronic undernourishment was already supposed by Elliot. Peretz (1929,361) found many chronic cases of undernourishment among his glaucoma patients. In experimental animals Küchle (1958,217) found that on feeding with a cholesterol-rich diet the incrustation with lipids of eye tissues (e.g. arcus senilis) and of vessel walls in other body organs, could be hindered by supplements of vitamins A and E. It seems possible, that the early aging in hot countries is related to the generally low intake of vitamin A. Another possible way in which nutritional factors can influence the development of glaucoma, is by the ingestion of toxic substances, e.g. mustard oil, which if accidentally mixed with Agremone mexicana, sometimes causes epidemics of glaucoma in Bengal (epidemic dropsy, Maynard, 1908,279 ; Lal and Roy, 1937, 210). One of its components, Sanguinarine, was first thought to be the toxic element, but Dobbie et al. (1961,90) in experimental animals found only a passing influence on intraocular tension, whereas Rodger et al. (1960,396) found that another component, citral, had no influence at all on the system of intraocular tension stabilization.

Heredity. Waardenburg from his own cases and from the literature estimated that Glaucoma follows a pattern of simple dominancy in certain families (1932,541) and that it is not necessarily the same type of glaucoma which is seen in siblings, but that chronic intermittent subacute glaucoma can run in the same family (542). Lauterstein (1965,225) never found a case of Glaucoma among Yemenite Jewish immgrants in Israel, possibly by their lacking the hereditary trait. In Iceland (Bjornsson 27) where there is a very high glaucoma frequency among the old, there is much consanguinity.

In Indonesia, where studies of heredity are difficult by the lack of patronymous names and variable family ties, we still found a few" glaucoma families" In the Chinese, where patronymous names are in use and family ties are more stable, we saw even the known phenomenon of anticipation in glaucoma, where the younger generation develops glaucoma at an earlier age than the parents.

Elliot in South India often saw the combination of Glaucoma with small size of the bulbus, attributing this also to a common hereditary factor.

General disease. According to Weinstein (l.c.) there are few general diseases which are connected with glaucoma, except essential hypertension and arteriosclerosis.

In the older literature often Syphilis is considered to be a contributing factor to glaucoma. Coppinger (1927, India 62) saw positive tests in ca 30% of his glaucoma cases, and insists that surgery can only take place successfully after specific treatment. Luque (1929, 246) in Chile found even higher figures: 80% of his glaucoma patients under 40 years of age had Syphilis, 48% of the middle-aged, and a lower percentage in older people. The non-syphilitic ones mostly had arterioscerosis, according to him. From his description it seems primary glaucoma is concerned, not glaucoma secondary to e.g. uveitis.

Knox (1961 208) observed glaucoma among 164 patients with rests of interstitial keratitis, mostly aged around 35 years. Sugar (1962 490) describes 4 similar cases, in which the corneal diameter was small and the anterior chamber shallow. Britten & Palmer (1964 42) twice saw similar cases.

In the classified lists of causes of blindness in the previous chapters, sometimes the etiological number 19 (syphilis) is given to glaucoma cases, instead of the usual 91 (unknown to science) to show the number of cases where we found the combination of glaucoma and syphilis. Routine Wassermann tests were not done on all glaucoma cases at Undaan.

Eye diseases.

It has long been thought, that Trachoma enhances the risk of developing glaucoma. The theory is based on the longterm inflammatory process in the limbal region, which possibly causes damage to the structures around Schlemm's canal. Burns of the cornea, especially those in the limbal area can cause ocular hypertension (Kümmel, 1915, cited by Elliot 105) and in analogy herewith it is argued that longstanding pannus and possibly also Herbert's pits in Trachoma can cause glaucoma. Cuénod & Nataf (1928, 68) suggest this, to which Terson (ibidem 68) in the discussion adds that possibly treatment of Trachoma with peritomy has the same effect. Bargy (1929, 20) in Indo-China doubts if Trachoma is an important etiological factor in glaucoma, because only 25% of the blind glaucoma patients showed signs of Trachoma ; he saw more often Syphilis. Peretz (1929, 361) in Egypt found traces of Trachoma in 87% of his glaucoma cases, but he does not mention how high this percentage in the population at large is (which could be high in Egypt). Wilson (1939, cited in Eisa Hamsah 102) feels that the high frequency of glaucoma in Egypt is to be ascribed to Trachoma, but Meyerhof (ibidem 102) does not believe so. Nema et al. (1964, 318) in India evaluated 225 trachomatous eyes and 82 non-trachomatous ones, and found that the eyes with Trachoma had a higher mean ocular tension, a decreased number of aqueous veins, narrower chamber angle, heavier trabecular pigmentation and higher tendency to goniosynechiae. All of these factors could cause lowered aqueous outflow.

Corticosteroids.

These drugs which are in this country often prescribed by general physicians on insufficient indication, because they can make a red eye pale in a short time, can locally applied cause an increase in ocular tension and field losses in cases of unsuspected wide angle glaucoma. Pasmanik & Miranda (1965, 355) found this in 56% of their cases, Nordmann et al. (1965 325) even in 90%. If the corticosteroids are stopped in time, the changes are reversible, but this can take a month or even more. In families of glaucoma patients the local application of corticosteroids is even used as a provocative test to discover subclinical cases in siblings. Because of the indiscriminate prescribing of these drugs there is a lot of corticosteroid ointment and eyedrops loose among the population. Patients tend to give the leftovers to friends and relations to let them enjoy the effect of the miracle drug. Corticosteroids have a very strict indication, i.e. certain specified corneal affections, and iritis and iridocyclitis. Contra-indications are : Trachoma, Glaucoma, herpetic corneal affections. As the latter are more frequent here than the former, a word of warning is certainly useful: general physicians should be wary in the use of local corticosteroids, and not listen too obediently to pharmaceutical representatives who are often not sufficiently impressed by the risk, as they like to sell the expensive drugs.

Aging.

Glaucoma has for a long time been placed in the group of diseases of the eye caused by "aging", along with senile cataract, senile macular degeneration and such ocular affections as those caused by hypertension and arteriosclerosis and diabetes. (Jayle, 1958, 188). Leydhecker (1985 233) stresses the point that among se-called healthy eyes there is subclinical glaucoma to be found even in the 3rd decade of life, whereas others (1963 362) ascribe glaucoma at a young age to late congenital or juvenile glaucoma. If Leydhecker's percentages per decade (see above) are plotted in logarithms the resulting curve is a straight line, like other senile eye affections investigated by Fischer (1948 121) do. The latter considers this a characteristic of "saturation" as in chemical processes, and concludes that aging in the eye is dependent not on historical time, but on biological time, This could quite well apply to glaucoma also and especially in this country.

On the physiological side Francois (1958, 127) states that by some investigators (Goldmann, 1951 Weekers et al. 1956) a decrease in the outflow facility was found with age, although others among them Granit, 1950 did not find this.

Anatomically there have been found signs of "aging" in the whole apparatus involved in the stabilization of the ocular tension (iris, ciliary body, chamber angle, choroid, sclera) which could easily lead to a slight or a serious imbalance between production of aqueous and outflow, causing subclinical or manifest glaucoma.

In this country other signs of early aging have been found by Han Tjwan Poen & Zainal Arifin (1965, 166) in the aorta wall. Possibly in connection with Küchle's finding of the preventive effect of vitamin A and E of "aging" (see above) it could be supposed that the general low intake of these vitamins in Indonesia has something to do with the early aging generally and consequently with the early emergence of glaucoma. Collaborating factors could be longstanding external eye infections, the stress of hard work in a hot climate, and possibly the high pigment content of the eyes, which on dispersal can lead to obstruction of the route of outflow of the aqueous. Heredity and hormone imbalance in women in and around the climacterium are to be taken into account although precise data in this country are not yet at hand.

Prevention of Glaucoma.

Prevention of Glaucoma as a disease of aging is impossible, but against the serious sequelae several measure can be taken:

I. In an effort to postpone aging, propaganda for more adequate feeding habits would be useful.

2. Contributing factors, e.g. Trachoma, Syphilis, should be treated early and effectively, still better : be eradicated.

3. Surgery of mature cataract, also if only monocular, should be considered especially in patients who live far from ophthalmological help.

4. All types of medical personnel, from doctors to midwives, should be alert to the possiblility of developing Glaucoma in older people.

5. General physicians should be very careful in prescribing local cortico-steroids for the eye, adhering strictly to the indications and contra-indications.

6. Eye doctors should conscientiousy use their tonometers in all patients over 35 years of age, whether they come for reading glasses only, or not.

7. Eyedoctors should consider favorably the advisability of preventive surgery for the "healthy" eye in cases of as yet monocular Glaucoma.

8. A drive for early detection of Glaucoma by surveys of whole population groups would be useful, but cannot yet be considered because of socioeconomic conditions.

Chapter Twenty two.

Some general infectious diseases sometimes causing blindness.

Morbilli. Measles.

In some tropical countries measles have a high mortality rate and rather often can cause blindness Probably the cause is a subclinical or latent vitamin A-deficiency, by which the usual conjunctivitis of measles is aggravated until the cornea is also involved, possibly even leading to Keratomalacia, on the other hand the vitamin A-deficiency will probably also have an adverse influence on the healing of the general disease, even causing a higher mortality than is usual in a well fed children's population. Aalbers (1941, 1) described this from Sumatra among Bataks, who probably aggravated the situation by their habit of giving feverish children cold showers without drying them afterwards. Xerophthalmia as a sign of general vitamin A-deficiency was prevalent in the region. Thygeson (1964, 511) mentions that in Africa (no region stated) Measles often result in blindness by their association with Xerophthalmia. Wessels and Johnson (1957, 560) describe from South Africa frequent ocular complications of measles, and suppose that a combination with Trachoma may be disastrous ; without explicitly mentioning Xerophthalmia, they state that many children in the region suffer from subclinical malnutrition "which is notoriously difficult to diagnose".

During the years covered by the present study we saw at least 5 children of whom the parents told us, that they had been suffering from Measles recently, or who were still sick. Mostly the eye disease took the form of Keratomalacia.

Variola, Smallpox.

In countries, where Variola is not yet under control, eye complications are not rare. Eugen Fick (1899 in Gräfe-Sãmisch Handbook 118) gives a good description of the eye complications, which is still valid. In the first few days the eyelids take part in the general eruption and the conjunctiva itself can also show the typical lesions. But the real danger for the eyesight is caused by the process in the cornea, which emerges after 2 to 3 weeks, in the form of a dense diffuse keratitis, and which can be a protracted process. Westhoff (1910 561) also stresses the beginning of the serious corneal involvement after the smallpox themselves have more or less healed. Like Fick he accuses relative immunity which is built up by the previous infection, but which develops slower in the avascular cornea. Fleck (1921, 124) also describes a case of disciform keratitis which started after the smallpox had healed. Mulock Houwer (1936. 303) saw in a few cases white spots in the iris after smallpox, indicating that the iris had been taking part in the disease. Voorhoeve (1937, 538) saw phthisis bulbi developing in a case of variola, which had been vaccinated. Basarodin and Heath examined the eyes of patients in Variola wards at Jakarta

(22) and found corneal lesions in 7.7% of adult patients, in 3.5% of the children, besides various palpebral and conjunctival lesions. They ascribe this favorable result to energetic antibiotic treatment. It is not clear if the patients were counted who possibly developed corneal involvement after healing of the smallpox, after dismissal from the Variola ward.

In the period under investigation 1958—62 we saw 15 patients in all who were blind by Variola, and a number who had been blinded in one eye only. A few had iris complications, and most of the cases had been healed long ago.

During the last months of 1964 and the first half of 1965 we saw many fresh cases of eye involvement in Smallpox at Undaan: 8 patients had corneal ulcers more or less simultaneous with the general disease; 40 showed the typical diffuse keratitis, 2—4 weeks after the general eruption, 3 of them showed a hypopyon. One of the children had indolent arthritis and periostitis of the legs, which healed after ca. one month. This complication is exactly like the description in Kouwenaars book on tropical diseases (213) p. 247. 2 children had vaccinia pustules on the eyelids, and 4 children had the same type of diffuse keratitis as seen in variola, but according to the parents thay had been suffering from varicellae. During the same period of almost one year's time we saw 6 cases of sequelae of smallpox of long standing, and 2 cases which had been probably blinded by smallpox. After that time the epidemic subsided, and for the last few years smallpox with eye complications was again rare, as is usual in this region. The patients we saw were not examined while in a variola ward like the cases in Jakarta, but they had been there in a few cases and came to us afterwards, as their eye complications emerged.

In the list of causes of blindness of Oey Khoen Lian and Maäs, variola is not mentioned, Mrs. Go-Sennema at Bandung saw one case in a year's time. Van Manen (1938. 262.) saw 12 persons blind by smallpox.

Mathur and Mathur (274) saw a case of optic atrophy after smallpox. In our lists 2 such cases are included for 1958—62.

Tuberculosis.

Tuberculosis can cause blindness by involvement of the : cornea, uvea, retina and optic nerve. Especially in children the type of tuberculous disease, which is known as scrophulosis can cause serious impairment of visual acuity. In prewar times in Indonesia it was the general opinion that true scrophulous keratoconjunctivitis was much rarer here than in Europe, and oftener seen in adults than was usually the case in European countries. There was found a parallel in the impression that here also surgical tuberculosis and scrofuloderma were more often a disease of adults than of children, contrary to what was usual at the time in colder climates (Soetomo. 462.). Before and including World War I keratoconjunctivitis scrophulosa had been very common in many European countries, e.g. Sämisch saw in Bonn that 21.4% of all outpatients had scrophulosis, Bertram, Düsseldorff, 1891 saw 45% and 1897 still 40%. Straub (1912) said that "the largest part of eye patients are scrophulous". After the first World War this figure decreased sharply (Rochat, 1932, 394) although tuberculosis in general not yet decreased in the same tempo. In 1939 in Groningen scrophulous eye disease was rare, but during the war and occupation years it increased again, which we ascribed at the time to lack of soap, lack of appropriate food (fat and proteins) and which could be cured even without the use of antibiotics which were unknown, by cod liver oil, good food and cleanliness, kept up for several months.

In Indonesia the issue of scrophulous ophthalmia has been obscured by the prevalence of "phlyctenules" in many types of conjunctival infections, and also in Xerophthalmia. These are far more frequent than the true scrophulous phlyctenules. Because of this many of the lists of diagnoses published cannot be used for assessing the frequency of scrophulous eye disease, as both types of phluctenules are mixed. More to be trusted are figures for specific keratitis fascicularis, which was found by Wille (1923, 572) at Semarang 1913—1920 in 5 cases, by Hötte (1928 179/180) in the year 1927 in 8 cases. Soewarno and Erwinn (cited by Soetomo 462) stated that true scrophulous keratoconjunctivitis was not frequent, as was also said by Bakker (1927, 17) and de Langen and Lichtenstein (222.). Sie Boen Lian (1933. 436) who wrote a thesis on the subject showed that the phlyctenes as seen in various kinds of conjunctivitis, and those seen in true scrophulosis were pathological-anatomically the same, and feels that the phlyctenules as such are an indication of some type of allergy, which in colder climates tends to be tuberculous, and in tropical countries could be caused e.g. by helminthic infestation of the intestines. He found that scrophulous eye disease is commoner in children, whereas the other type of phlyctenules are as often seen in adults.

From the Philippines scrophulous keratitis has always been accused to be an important cause of blindness, and Agarwal (4.) describes the disease from India. During the period of our present study we saw only one child blinded by this cause, but slight visual impairment was rather more often seen in children, who showed the typical habitus and eye signs of scrophulosis : blepharospasm, fascicular ulcers, phlyctenular keratitis, otitis media, rhinitis, facial eczema, and in most cases a positive Mantoux test. Cervical glands were very common in these cases.

As a factor in causing Xerophthalmia, tuberculosis is rather often found (see chapter on Xeropphthalmia).

While in the thirties scrophulosis was rather rare in N.W. Europe, other eye affections caused by tuberculosis were not, especially those of the uvea. In Indonesia syphilis has always been a far more important cause of iritis etc. but in prewar times there were a few case presentations of tuberculous uveitis (Fast, 1936, 112. Soemitro Hadibroto 1935, 459) and Eales' disease (Leber 227).

In the period of the present study we saw a few cases of tuberculous uveitis, but many more of syphilitic uveitis.

A "modern" cause of blindness by tuberculosis is that caused by optic atrophy in healed tuberculous meningitis. Before the advent of antibiotic therapy this disease was always fatal, but according to Mooney (1956, 290) 80% of cases now survives, unfortunately 20% of them blind by optic atrophy. In this country this is not yet a common cause of blindness in children, but it seems to be on the increase in recent years.

In recent times Gat (1963. 138) feels that in Hungary phlyctenules are still to be considered a cause of alarm, as 38% of his patient material with phlyctenules had an active tuberculosis, and curiously the same goes for Indonesia, where Nana Wyana at Jakarta, 1960—63 found signs of tuberculosis in 36.61% of phlyctenulous patients (89).

Leprosy.

Blindness in leprosy can be caused by: iridocyclitis which ends in phthisis bulbi; lagophthalmos (caused by involvement of the facial nerve) leading to keratitis e lagophthalmo, and less often by leprous involvement of the cornea it self.

According to Louwerier (Sumatra, 1923, 245) and Bakker (1932, 18) Leprosy is rare as a cause of blindness in Indonesia. Wille (1923. 572) mentions in his list of diagnoses of outpatients for 1913—1920 at Semarang 6 x corneal leprosy and 6x irisinvolvement (how many were blinded is not stated). van Manen (1938, 262.) saw 2 cases of blindness by leprosy, in the lists of Oey, Maas, Go-Sennema it does not figure at all.

In the lists of the present study 4 cases of blindness by leprosy are found. Treatment of leprosy is much more hopeful than it was formerly by the discovery of modern antileprous drugs. For local treatment of the eye corticosteroids are of value. R.G. Cochrane and T.F. Davey, in their "Leprosy in theory and practice" (1964, 59) 321 state "that leprosy blindness should be very rare and long delayed if the signs are recognized in time and if appropriate measures are undertaken".

Febris typhoidea Typhoid fever.

In rare cases typhoid can cause optic atrophy, which was decribed by Oey Khoen Lian at Jakarta (1940, 330) and which we saw in one case during the 1958—1962 period.

Chapter Twenty three. Miscellaneous less important causes of blindness.

Sympathetic ophthalmia. Many authors have commented on the relative rarity of sympathetic ophthalmia in colored races (Ida Mann: "Regional distribution of eye diseases" 'AJO'74/5,II: 138.1959; Somerset, 1963,466). Evans (1963,108) from Rhodesia states that in spite of many perforating eye accidents the complication is rare, supposing that if a virus infection is the cause, this possibly is more virulent in colder climates. Khosla et al. (1965,203) saw a case after iridencleisis, whereas Tomar (1965,514) also from India, never saw a case from 1948-1953 in 140 cases of perforationg eye injuries. Schubert (1964,420) saw 2 persons who were presumably blind by sympathetic ophthalmia in 87 blind persons (not histologically verified).

In Indonesia Mulock Houwer (1936, 299) gave a case presentation of one case, mentioning the apparent rarity in Indonesia, and saying that most cases are caused by perforation after conjunctivitis gonorrhoica or ulcus serpens. Van der Horst (1939, 177) showed 2 cases, both after perforating injury, and Leber in the discussion on this case, stated that the complication is rarer on Java and also less malignant than in Europe. Yap Hong Tjoen (1941; 592, 593) demonstrated a case where sympathetic ophthalmia struck the second eye two weeks after enucleation of the injured eye, which had been removed three weeks after the accident. In 1939 the same author showed one case of fully developed sympathetic ophthalmia, and at the same time two others who were endangered.

In the classified lists from Undaan Hospital there are to be found: 1 case in a boy, 3 in men under 40, one in a women under 40, one in a women over 40 years, all of them after an accident, and one in a woman over 40 after glaucoma surgery. The complication seems not to be so very rare, and it must certainly be kept in mind in a careful follow-up of all eye accidents, including surgery. Enucleation is often refused, but treatment with corticosteroids seems promising : a trial with these drugs in case of refusal of enucleation must be made, and sometimes it happens that the final visual acuity of the first eye is better than that of the second, sympathizing, eye.

Congenital malformations. In countries where the prevention of infectious and nutritional causes of blindness has been effective, the remaining blinding diseases consist for a large part of congenital malformations and hereditary forms of blindness, among them those caused by transmitted maternal infections (Toxoplasma, Syphilis and Rubella, possibly other viral infection). A stringent diagnosis of Toxoplasmosis cannot yet be made in Indonesia for lack of opportunity to perform the necessary tests. Congenital Syphilis is already considered in the chapter on Syphilis, Rubella in the chapter on Cataract.

Congenital malformations caused either by vitamin A-deficiency of the mother (Jackson and Kinsey, 1946, 187; Warkany, 1954, 547) or by vitamin A-intoxication of the mother (Baba and Kotani, 1959, 11) have so far only been produced in experimental animals.

In Indonesia congenital causes of blindness so far are still only a small percentage of the total causes of blindness. Malformations run from total anophthalmos, of which Bais (1937, 16) showed a case to minor affections like "blue sclera", of which Zuidema (GTvNI 1941 : 1993) showed a case in a Javanese girl, where no other cases were found in the family. We saw the syndrome in a few Indonesian and Chinese families. Soejoenoes (1941, 457) saw a case of Sturge-Weber syndrome. Albinism is found especially in some families hailing from the Minahasa (North Celebes) but also in others.

Retinoblastoma has been assigned a special chapter because of its importance, the next important congenital/hereditary affection is **Retinitis pigmentosa.** Recently some interesting points have been investigated relating to this affection : Chatzinoff et al. (1958, 55) supposed that the special 11-cis-isomer of retinene was probably lacking in these patients and instituted therapy on this basis. Campbell et al. (1965, 49) found serum levels of vitamin A to be low, and Shearer (1965, 427) saw that absorption of beta-carotene was impaired. Treatment of retinitis pigmentosa so far has been a crux medicorum where almost everything has been tried, and often at first hailed as successful, because of the spontaneous remissions which in this disease are not rare, and later mostly admitted to be yet another disappointment. Therefore these findings which could give new pointers to treatment on a rational basis are welcome. Dowling, in analogy with a hereditary type of blindness in rats, which looks like retinitis pigmentosa and which could be retarded by keeping the animals in the dark, suggests that persons at risk should use dark glasses.

In our classified lists we found : 2 boys, one girl, 15 men under 40,10 women under 40, 13 men over 40 and 7 women over 40 years already blinded by Retinitis pigmentosa.

We also found a few cases of **high Myopia** on a familial basis, as well as familial glaucoma. The closer investigation of many of these familial and hereditary diseases is very difficult because of a complex of social causes, making it possible that e.g. among our patients with optic atrophy e causa ignota are hidden cases of Leber's optic atrophy. Mulock Houwer (1940, 309) guessed at this possibility in one of his cases.

Diabetes. Diabetes plays an increasingly important role as a cause of blindness in countries where life expectancy is high and treatment of diabetes efficient. It is especially dangerous in young persons : Berkow et al. (1965, 24) describe a group of blind persons who had received a guide dog. From 180 of them who were blind by diabetes, the average age of onset of diabetes was 14.6 years, at onset of severe blindness 31.4 years (retinopathy). The average lifespan after the onset of blindness was only 5.8 years. Sorsby (1966, 472) gives a percentages of 7.1% for all ages in England, Schappert-Kimmyser (1959, 414) 3% of all ages in Holland. Decaudin (1952, 78) stated that 27% of chronic diabetics lost their visual working ability.

At Undaan we found only one women under 40, and one man over 40 years who were blind by diabetic retinopathy, whereas diabetic cataract was somewhat oftener seen.

From Indonesia Zuidema described a form of juvenile diabetes with pancreatic calcifications, complicated by cataract (1940, 604 ; 1955, 605). Ratnaike et al. (1963, 389) report on the same syndrome from Ceylon, but instead of cataracts which are not mentioned, the patient showed long eyelashes and eyebrows.

In the classified lists from Undaan hospital we find 2 men under 40, 2 women under forty, 6 men over 40 and 2 women over 40 years with cataracts and diabetes. All cases of cataract in the ouptatients' department are tested for glucosuria on the first examination, and in 1960 we found glucosuria in 15 of 450 cataract cases. Until now we never recognized the syndrome of Zuidema. Surgery should be performed only after the diabetes is under control, but unfortunately this presents difficulties, i.a. because people are careless, and have the idea that diabetes can be "cured", for which the criterium is a (temporary) absence of glucosuria. Cases where cataract surgery is disappointing because of existing retinopathy can happen.

Retinal detachment. Some authors comment on their impression, that retinal detachment seems to be less frequent in pigmented races than in white people. Brown and Thomas (1965, 43) give some figures from which can be concluded, that primary detachment is more frequent among white Americans than among American Negroes, whereas detachment after cataract surgery was not encountered in Negroes altogether.

In Indonesia where retinal detachment in prewar years was a well-known picture, which was treated surgically, Mulock Houwer (1939, 308) feels that it was rarer than in Europe. My impression is the same, although we saw a few cases, and after cataract surgery in one girl and in one man under 40 yrs. retinal detachment developed.

Ulcus rodens corneae (Mooren). According to Harley (AJO, 44/1 : 121-122, 1957) this affection is so rare that it was unusual for one ophthalmologist to have reported more than 3 cases of his own. Mulock Houwer (1937, 306) stated that on Java Ulcus Mooren was far more frequent than in Europe. We saw several cases, certainly more than 3 per doctor, mostly in women who looked prematurely aging, but also in younger patients. Kuriakose (AJO, 55/5 : 1064—1069, 1963) from India claimed that almost all his cases had Ankylostomiasis, which should be treated. We tried this out in several cases, finding that the corneal disease in the Ankylostoma-positive cases reacted favorably to specific treatment, but cases without ankylostoma we treated by admission to hospital, which gave them better food and more rest than they were used to, and various kinds of local treatment (diathermy, conjunctival bridge covering etc.), were often disappointing.

Acquired tumours of the eye. Apart from retinoblastoma, blinding eye tumours in Indonesia are rare. They consist in other climes mostly in the feared melanosarcoma uveae: Bakker (1932, 19) mentions cases of this tumour, but does not state the race of the patients. Mulock Houwer (1935, 301) went over all specimens of eye tumours preserved in the Cancer Institute and over the cases mentioned in the literature and could find not one certain case of this type of tumour in a Native Indonesian. The rarity is also commented on from China (Fuchs, 1962, 132) and Egypt (47). Soetamsi Sastroroedjito (1937, 461) published one case of a patient who had a phthisis bulbi and liver tumour. Post mortem both showed melanosarcoma. In a much earlier stage enucleation of the eye had been refused.

In over 20 years I saw only one case of this tumour in a half-caste young man. The rule-of-thumb that a painful blind eye of uncertain diagnosis should be removed, as the danger of a melanoblastoma is always present, is mostly not adhered to in this country, in view of the rarity of the tumour and the aversion against enucleation in most patients.

Another pigmented tumour can develop from the very frequently seen pigmented nevi of the conjunctiva, which are not all of them as harmless as Ida Mann (”regional distribution of eye disease”, AJO 47/5, II : p. 139, 1959) supposes. Mohamad (1937, 288) found a fatal case of melanoma of the conjunctiva with extensive metastases. Melanine in urine was positive. We found rare cases also, where a nevus turned malignant. In spite of their very common occurrence they should be regarded with some suspicion.

Neuropticomyelitis (Dévic). In this disease initially the pupils do not react to light, although the fundus is normal. Afterwards optic atrophy is found. Verhaart (1937, 537) showed a single case in Indonesia. We never recognized the disease but it is possible that it is hidden in the group ”optic atrophy e causa ignota”.

Blindness by snakebite. The two large groups of snake venom, one haemotoxic, the other neurotoxic, can both of them cause blindness. Our only patient had extensive retinal hemorrhages, also situated centrally which damaged his vision permanently. The haemotoxic kind was seen by Guttman Friedmann (1956, 153) in Haifa, causing optic neuritis like in serious hemorrhagic anemia. Davenport and Budden (1953, 76) described a case from Nigeria, where spasm of retinal vessels was the cause of blindness. Treatment: cortiocosteroids. (54)

Chinine blindness. Cases of chinine poisoning can occur if children have access to the drug, in cases of suicide (Mulock Houwer, 1941, 315) and in induced abortion. The primary damage concerns the visual elements and the ganglion cells, after a few hours extreme narrowing of retinal vessels and a white disc can be found. In non-lethal cases after a few days visual acuity is often restored somewhat, but retraction of the visual fields and nightblindness will ensue often, also in favorable cases.

Chapter Twenty for.

Xerophthalmia.

Motto: "It is probably true to say that the enormity of the problem of infant xerophthalmia is only apparent to those who have at some time or another had to witness the hopeless procession of human tragedy through hospital wards and out-patient departments. When the cause is known, prevention and early cure cheap and effective, and yet the flow of incurable cases is undiminished year by year, it is little to be wondered at that the attitude of the observer may become cynical when the academic achievements of western medicine are seen to be powerless in practice" (Mac Laren, 1963).

In agreement with modern usage as proposed by Oomen (348) and Mac Laren (256) the term Xerephthalmia is applied to all eye affections which have to be attributed to vitamin A-deficiency in the body. The type of "xerosis bulbi" seen in cases of inveterate Trachoma and other chronic eye diseases causing drying of conjunctiva and cornea, is NOT included.

The word Xerophthalmia means "disease of the dry eye" (NOT thickening of the conjunctiva as is stated in some texbooks; Greek "xeros" means: dry). As the aspect of the eye really is dry, the term is adequate. I include also nightblindness if it is clearly of the same etiology, i.e. caused by vitamin A-deficiency. In accordance with McLaren I avoid the term" hemeralopia" (which on the European continent means nightblindness, in Anglo-Saxon countries: dayblindness) and "nyctalopia" (which on the continent means dayblindness, and in Anglo-Saxon usage: nightblindness). The misunderstanding of these terms according to Hirschberg (173) is of a venerable age: Hippocrates used the word hemer alopia for nightblindness, and was supposedly misunderstood by Galen, who thought that nyctalopia meant nightblindness. Misunderstandings of over 2000 years' standing are not amenable to correction, so to avoid further complications it seems advisable to use a term which is self evident, i.e. nightblindness. In Indonesian, Literally: buta malam, among the populace also known as: buta ajam (blind like a chicken) or: Rabun sendja (twilight blindness).

Extensive literature as well on vitamin A itself (Moore, 1957,291) as on Xerophthalmia (Oomen, Outline of Xeropthalmia, 1961, 348, McLaren, Malnutrition and the Eye, 1963,256, WHO Reports, 565,569, 570) is available, whereas Teng Khoen Hing (504,505,506) writing on fundus lesions in Xerophthalmia, gives a review of much of the literature as it appeared in Indonesia and Holland. Therefore I can limit the exposition of the findings of others to essentials and to some points which seemed interesting in connection with my own study. In conventional textbooks of ophthalmology there is very little to be found on the whole field related to vitamin A deficiency as it is seen clinically, but of the function of vitamin A as related to vision there is already an extensive body of knowledge.

Dual function of vitamin A.

Vitamin A is absolutely essential to the function of the retina in seeing, but it also plays an important role in the rest of the body although it is as yet not precisely known which role.

1. Function of vitamin A in vision.

After Holm in 1925 had discovered that rats on a diet insufficient in vitamin A became nightblind, Yudkin (597) saw that vitamin must be present in the retina itself, and Wald (1935) showed that vitamin A as such takes part in the cycle wherein rhodopsin in the visual rods is bleached by light, and reformed on dark-adaptation. In 1958 Dowling and Wald described the series of events which is instigated by diet insufficient in vitamin A: first depletion of liverstores of the vitamin, next lowering of the vitamin A level in the blood serum, followed by decrease of rhodopsin content of the retina: at this point nightblindness appears, followed by other signs of deficiency ; lastly destruction of visual cells in the retina. According to Wald the rhodopsin in the rods and cones plays an integral part in their structure: it seems that the two parts (retinene: vitamin A-aldehyde, and opsin : the protein part) are only stable if both are present in appropriate quatities: if one decreases the other will also break down. Rhodopsin makes up a large part of the rods and cones, although the vitamin A component itself is only ca. 1% of its weight.

Anatomically the outer parts of the rods are made up of flattened vesicles, which look like rolls of coins. It is possible that if one of them is bleached by light, the signal will be amplified by the others and transferred to the nerve cell connected with it (Missotten, 287; Lieb & Knauf, 1964, KMA 145/5). By the special arrangement of synapses between the ganglion cells in the retina it is possible that a signal from more than one rod or cone is received by them (Peckham, 360), influencing the strength and local sense of the light impulse. The present methods of study include electron microscopy and electroretinography. With the latter method it has been possible to find early signs of lowered dark adaptation in the retina of animals, and also of children, as they make objective measuring of dark adaptation possible. Methods which depend on intelligent cooperation of the patient are less reliable.

Another objective method of measuring visual acuity and light sense makes use of the opticokinetic reflex.

Bairati and Orzalesi (1963,15) found that in the human retina the pigmentepithelium is organized like a filtering epithelium, probably related to the metabolism of the photoreceptors.

Chemistry. The chromoprotein rhodopsin is composed of opsin and 11-cis-retinene. By light this is isomerized to all-trans-isomers, which do not form a stable chromoprotein with opsin, thus forming a mixture of all-trans-isomers and opsin (Hubbard, 183). The first step of this transition produces the visual excitation (from rhodopsin to lumi — or at most to metarhodopsin). Regeneration can only take place if there is present opsin and 11-cis-retinene (Wald, 544). In vivo this process goes slower than in vitro, because of necessary preceding other processes in the rods and cones. The spectrum of rhodopsin in rods agrees well with the spectral sensitivity of human rod vision measured at the color of the visual pigment is probably not caused by at race metal (Fukami et al., 1959,133). Wulff et al. (1958, 582) showed that flash illuminated rhodopsin in solution is changed into a mixture of retinene isomers bound to protein. Koide (1957,210) stated that by providing an energy supply by glycolysis the regeneration of rhodopsin is accelerated, whereas Wald thought that no energy was needed for the regeneration. Rushton and Campbell (1962,404) developed a method whereby the amount of rhodopsin in the retina can be measured in the living human eye by the amount of reflexion of a blue-green light. It appeared that rods are visually inoperative with less than 90% of their rhodopsin present. By the equivalent of a clear sunny day rhodopsin was not more than half bleached. Weale (1962,551) found with the same method visual pigment different from rhodopsin. Gloster and Greaves (1964,142) used the method with different colors, identifying different color receptors.

The breakdown of rhodopsin in vitro into opsin and retinene takes place at the SH-bond, whereby the number of SH groups is increased (Wald). Cernoch and Dolenek (51) found this also to be true in retina homogenates.

Vitamin A is an alcohol, which according to Andrews and Futterman is converted into its ester during light adaptation in the retinal microsomes, after having been oxydized in the outer segments of the retinal visual cells (8. 1964,135). By direct microspectrophotometry single cones of human retinas could be examined, making it probable that there exist different kinds of color receptors, probably three, with different visual pigments (Wald, 1964, 545 ; Marks et al. 1964,271)

Mc Farland and Munz (253) showed in 2 kinds of freshwaterfish that the visual pigments differed because of a different protein. Hybrids of the two kinds of fish had visual pigments which behaved genetically like a singlefactor difference, making it probable that the differing absorption spectra of visual pigments are related to substitutions of amino acids in the protein parts.

Electroretiograpihcally Cone and Ebery (1965,60) have found that the two waves in the ERG, one arising in the receptors, the other in the bipolar cells, already identified by Noell (324) are independent of one another, which implies a division of the visual excitation into two different signals. Dowling (1967,96) found that the wave arising in the bipolar cells shows typical adaptation properties, on light adaptation, whereas the wave arising in the receptors fails to do so. Pak and Ebery (1965, 353) by observing at low temperature where some steps in the bleaching process of rhodopsin are known to be stable, concluded that the positive peak of the early receptor potential is occurring after photo-isomerization of rhodopsin, but before metarhodopsin has decayed into retinene and opsin.

Pathology. Koyanagi (1920,214) could observe a case of hemeralopia which had been in existence for one month, complicating liver disease of 3 years' duration. He found granules in the retinal pigment epithelium, in its outer layers, regularly divided from optic nerve to ora serrata, staining yellow red in Sudan III. Ibata (1930,185) found less lipoids in outer limbs of rods and cones of albino rats who had been fed a vitamin A-free diet. Ida Mann et al. (1946,264) saw in the retina of rabbits, who had been fed a diet with varying degrees of vitamininsufficiency for varying times, changes in the retina ranging from complete split of outer and inner limb of the visual cells to no change at all. Rodger et al. (398) first fed Macacus rhesus a vitamin A-rich diet, until liver stores were large enough, then made animals nightblind by a vitamin A-freediet: the conjunctiva and cornea remained intact in this way, the retinal pigment epithelium showed only a regrouping of pigment, which retreated into the cellbody, covering the nuclei. After Dowling and Wald had found, that retinoic acid can fulfil all functions of vitamin A in the body with the exception of the building of rhodopsin, it became possible to observe changes in the retina by vitamin A-deficiency without the animals otherwise showing any changes: Dowling (95) showed that especially the outer segments of the visual cells were swollen with segmenting of the disks, eventually ending in total disappearance. If vitamin A was given in time, the process could be reversed and regeneration took place. This was partially confirmed by Leeman (1962, 228) who saw also changes in the inner parts of the visual cells. Dunagin et al. (1964,99) identified retinoic acid as a definite intermediate of vitamin A-aldehyde metabolism, in the rat in vivo. Pitt (1964,375) states that probably the acid is the form of vitamin A that is active systematically (as opposed to in the retina) as it causes signs of vitamin A-intoxication sooner than the vitamin itself. In the cat, Scott and Greaves (1964,422) and in a lizard Eakin (1964,101) showed the same type of degeneration as in the rat (the lizard also in its light sensitive third eye).

All of these pathological-anatomical results make it probable, that vitamin A is not only needed for the visual function of rods and cones, but is to be considered as an essential part of their structure, which keeps the cell in shape by its stabilizing effect on its most important protein. Rhodopsin makes up more than 50% of the structure of the visual cell. Wald claims that this stabilizing function of vitamin A is not only active in rhodopsin but could well be active in other proteins in the body elsewhere as well. This is a relation which needs to be explored further.

Fundus xerophthalmicus. From Xerophthalmia found in liver disease, it has been known for a long time, that sometimes small white spots in the retina could be seen (Purtscher, 1900,384). From Japan these spot were described in nightblindness, and were differentiated from retinitis pigmentosa sine pigmento and from fundus albipunctatus, because the spots disappeared on the treatment with vitamin A. Teng Khoen Hing (1958, 1964,1965; 504,505,506) has made extensive studies of the affection which is seen at Bandung, Java, in rather large numbers. He first thought that it was an early sign of vitamin A-deficiency, but later on found it on the contrary in cases of protracted lowgrade vitamin A-deficiency. Pillat and A. Fuchs saw it in China between 1920-1930, but elsewhere either it has not been looked for or it is very rare. In Surabaja we found some cases which seemed typical. It is accompanied by night blindness and often by slight xerosis conjunctivae. When neither is present, doubt about the diagnosis is justified.

Metabolism of vitamin A in the body. Vitamin A itself is found only in animal foodstuffs. According to Glover and Walker (1964, 143) it is finely dispersed in the intestinal fluid of the upper part of the intestine, in mixed lipid micells, formed with bilesalts, lysolecithin, lower glycerides and cholesterol. Transport across the mucosal cells of the intestinal wall is thus facilitated. It is carried to the liver by way of the chylus system, where it is stored in the Kupffer cells as palmitate, and for a small proportion in the liver parenchym cells as alcohol. From there it is transported to the tissues as the need arises, as the ester form in a lipoprotein or as active alcohol in a specific protein carrier. Djaeni (thesis, Columbia university, 1964, cited by Poey Seng Hin in "Research in Indonesia, 1945—1965") confirmed the role of protein in the resorption of vitamin A, and found that the vitamin A acid uses the portal vein to reach the liver, whereas other forms of vitamin A go by way of the ductus thoracicus.

The body is able to extract vitamin A from provitamin A (carotinoids). The conversion takes place in the intestinal wall (Sexton et al., cited by Straub and Schmidt, 485) and the vitamin A thus formed follows the same route as described for original vitamin A. For the whole process is needed a certain amount of fat in the diet, especially for the resorption of carotene (McLaren, 256; Roels, Trout and Dujacquier, 1958, cited by: Roels, Madj. Kedokt. Indon. **11**: 417, 1961). For the transport of vitamin A in the blood stream probably also protein is needed (Arroyave and Scrimshaw, 1961, cited by Roels, 1. c.p. 418), whereas Platt (also cited by Roels) found that adequate liver storage of vitamin A also depends on sufficient protein in the diet.

Carotinoids are derived from vegetable sources, green leaves and yellow fruit. Their absorption and conversion into vitamin A is influenced by many factors : species differences, sex, cis-trans isomerism, disease, size of dosage, method of administration, are mentioned by Thompson (1965, 509).

If there is a disturbance in any of these multiple mechanisms which are involved in getting vitamin A to the retinal cells, the function of the latter will be damaged after a shorter or longer period of time, depending on the size of the liver stores at the beginning. Rodger et al. (1960, 396) found that other functions of the eye (stability of intraocular tension, outflow coefficient, capacity to regulate resistance to outflow) were not influenced by vitamin A in the rabbit.

2. **Function of vitamin A** in **other parts** of the body, except in the retina. A. **In epithelial cells.** In experimental depletion of vitamin A in animals and man, one of the common effects has been an alteration of epithelial cells. Wolbach (1938) found that epithelial cells, especially in transitional epithelium were atrophied (cornea, conjunctiva, bladder, ureter, calyx of the kidney, parts of respiratory tract and digestive tract, also in parotis and pancreas : Wolbach and Howe, 578 : Davies 77). Cells of the basal cell layer become hypertrophic, forming a continuous layer, whereas in glands and ducts there are foci of the new kind of epithelium which are not continuous. This layer which slowly lifts the original epithelium away, shows keratinization. On treatment with vitamin A the changes are reversible. On the conjunctiva the goblet cells atrophy, which are originally present in a large part of the conjunctiva, but the smallest number in the exposed part of the bulbar conjunctiva (Wolff, 579).

Yudkin (1924, 596) observed in rats on a vitamin A-deficient diet, corneal lesions starting in the deeper epithelial layers, with leucocyte infiltration, vascularization of the cornea from the limbus, leading to ulceration. The conjunctiva was xerotic, showing local keratinization. He felt this was comparable to keratomalacia in man. Mori (1924,292) found that the brownish discoloration often seen in Xerophthalmia, is caused by melanin, accumulating especially in the intercellular spaces of the basal layer of the conjunctival epithelium, not of the cornea. Kreiker (1929, 215) saw that also in apparently still normal conjunctiva in A-deficiency there are already changes in connective tissue, which has less fibers than normally. Pillat (1931, 1932, 1933 ; 368, 369, 371) describes this pigmentation extensively. He found it frequently in cases of longstanding A-deficiency in adults in North China. The pigmentation is seen in the inferior fornix, in the caruncle and in the exposed part of the bulbar conjunctiva, less in the upper fornix. According to Pillat it is seen in the white race also (Vienna, after World War I). He found the pigmentation to be produced by melanoblasts in the basal cell layer and also in the middle layers of epithelial cells. After treatment the normal desquamation accounts for the slow disappearance of the discoloration : there were no signs that the pigment was transported away into subepithelial tissue. Matsuoka (1932, 276) found the pigment in small quantities in subepithelial chromatophores and histiocytes and considers the pigmentation of the conjunctiva in A-deficiency to be an early sign. He could produce it in rabbits also. There appears no pigment in the cornea (277).

Ida Mann et al. (1946, 264) also examined rabbits on a vitamin A-deficient diet of varying duration, and found first a slight keratinization of the corneal epithelium, followed by pigmentmigration in the conjunctiva, then keratinization of conjunctiva and lacrimal glands, no vascularization of the cornea. The younger the animals, the sooner the alterations developed, in the mean after the vitamin A-level of the blood serum had fallen to ca. 10% of its original value. Treatment with vitamin A reversed all changes rapidly, except the conjunctival pigmentation and the absence of goblet cells.

Duke-Elder gives no information on the conjunctival pigmentation, only a few references.

Mellanby (1934, cited by McLaren, 256, p. 315) first thought that the eye changes in vitamin A deficiency were caused by damage to the Gasserian ganglion, and so considered them from the neurotrophic angle. Others could not confirm this, although the symmetry of the corneal lesions gives some support to the idea of a neurotrophic causation (Mulock Houwer, 297). Later Mellanby (cited by Maumenee, 279) showed in tissue culture experiments, that vitamin A in excess changes normal skin epithelium into mucous membrane-like epithelium, a change which is reversible by reducing the vitamin A excess back to normal. Hitomi (1936, 174) because of the lack of signs of infection in pathological specimens of Keratomalacia, also attributed the process to a neurotrophic factor.

B. **In the growth process.** It has been thought that vitamin A was a "growth vitamin", but it now seems more probable that it is needed for the best utilization of protein ("Protein-sparing" property of vitamin A, Roels, "A review of some recent work on vitamin A", Madj. Kedokt. Indon. XI : 412-419. 1961). In the experiments of Mann et al. (l.c.) on rabbits, the very first sign of vitamin A-deficiency was a growth retardation and from numerous other experiments (extensively reported in Moore, 1957, 291) there seems to be a close connection between vitamin A and growth. McLaren (1959, 256) gave young rats a diet very low in protein and vitamin A, keeping their body weight stationary. Although normally female animals develop xerophthalmia later than males, in this experiment the situation was reversed : the arrested growth probably needed less vitamin A than usual, saving the eyes from xerophthalmia ; male young animals grow quicker than females, causing them to use up more vitamin A for growing and leaving less for protecting them against xerophthalmia.

C. **In fat metabolism.** Küchle (1958, 217) found that vitamin A and E could in a number of cases retard the deposition of cholesterol in the cornea (arcus senilis) in animals which were fed a diet high in cholesterol.

Also the deposition in other organs and vessel walls was retarded by the combination of both vitamins.

D. **In corneal lesions.** Agarwal described a favorable influence on the healing of corneal ulcerations of vitamin A therapy, but McLaren found that slow healing of corneal wounds experimentally was only found if also protein deficiency was present (256). Nelken and Nelken (1966, 317) found that vascularization of the cornea after artificial trauma in animals was slowed by vitamin A locally and intraperitoneally. Fischer (1937, 119) attributed an unusually high number of cases of herpes corneae in men to lack of vitamin A in the diet of urban people on the dole. Sie Boen Lian (1938, "Hoornvliesaandoeningen en vitamin A", Hand. 8e Ned. Ind. Nat. Wet. congr.) found vitamin A levels in the blood of patients with corneal ulcers by gonococcus to be low, and recommended treatment with vitamin A.

Fischer (1949, 120) found in examinations with the reflectograph, that in incipient xerophthalmia the corneal epithelium is not wetted at all. The tear film breaks down instantly, not only after artificial opening of the eye. This indicates that the epithelial cells have undergone a change wherein probably the hydrophile groups in the most superficial layer are disarranged, causing a change in the surface tension.

E. **On offspring.** Jackson and Kinsey (1946, 187) reported on abnormal offspring of rats fed a vitamin A-defficient diet, but for the development of eye malformations the deficiency had to be so severe, that most litters suffered fetal resorption. Warkany (547) had found a possible connection with retrolental fibroplasia, but Jackson and Kinsey doubt the possibility. Baba and Kotani (1959, 11) on the other hand produced malformations in offspring of rats fed an excessively vitamin A-rich diet, viz. an-or microphthalmos.

F. **Vitamin A-intoxication.** Turtz and Turtz (1960, 519) found as signs of intoxication, after treatment of acne with 200,000 IU daily for $1^1/_2$ years!!: retinal hemorrhages and incipient papiledema. After treatment with vitamin A was discontinued the eye changes disappeared. W. Funder (1960, 134) found loss of eyelashes and blepharitis after excessive dosage with vitamin A and panthenol, applied to corneal lesions. How the various effects of lack of vitamin A or its overdosage are to be explained is not yet clear.

Eye signs possibly related to vitamin A- or protein-deficiency.

1. **Changes in eyelashes.** Normally there are 70-160 lashes in the upper lid, in 3-4 rows 8-12 mm long, and in the lower lid ca. 75 lashes, 6-8 mm long. (Bailliart, 14). Only in individuals over 10 years of age, Motegi (1931, 293) found lashes in the lacrimal part of the lid margin, i.e. nasally from the puncta lacrimalia. He also found more and longer lashes in Japan in colder parts of the country than in warmer regions. The lashes survive 5-6 months before dropping out. Avdylovitch (1960, 10) states that usually one third of the total number of lashes is growing, whereas 2/3 is "at rest". They will only drop out sooner than normal in blepharitis, when there are chemical changes in the sebum of the lid margin. Feer (1924,113) already has seen, that in Tuberculosis often "beautiful, thick eyelashes" are to be found. Shoji (1931, 431) describes these as well in scrofulosis as in pulmonary tuberculosis. Reiter (1926 and 1931, 387) found exceptionally long

eyelashes in a patient who on post mortem showed to have suffered from: metastases after surgery for breast cancer, atrophy of adrenal cortex and atresia of ovarial follicles. Maybe this was a Cushing-like syndrome as also growth of a beard is mentioned. Dik (cited by Mulock Houwer, 297) was struck by the length of eye lashes often encountered in Xerophthalmia, as was Oomen, who also drew attention to other changes: straightness, sparseness and irregularity of length, in Xerophthalmia as well as in Kwashiorkor. Rambo (1958, 387) from India mentions that often in the first stage of Xerophthalmia there are already long, thick lashes, but he does not mention if there were other deficiencies as well. Majewski (1958, 259) describes very long lashes in a 4 year-old child, otherwise healthy, stating that normally darkeyed persons have longer lashes than blonde ones, and that the length in children and adults is the same,i.e. average in upper lid 7.35 mm, in the lower lid 4.88 mm (100 persons measured). It seems not yet to be certain whether long eyelashes belong to vitamin A deficiency or to protein-calorie deficiency. Schöpfer (1929, 419) found increased length of the eyelashes in a case of keratitis neuroparalytica. Standard texts of ophthalmology do not mention the sign.

2. **Foam in the conjunctival sac and on the lid margin.** In Bitot's spots there can often be seen an aspect as if the conjunctiva is covered with foam. For the production of adhesive foam on the conjunctiva bulbi there is needed a slightly altered surface (keratinization) and probably also an altered composition of the precorneal film. The film covering the eye is composed of tears, meibom secretion products and epithelial debris from cornea and conjunctiva.

a. Tears. these contain normally: albumin, alpha 2-globulins,gamma globulins and lysozyme. The protein content is very low. According to Liotet and Reveilleau (1965, 242) in some eye diseases there is an increase of albumin and decrease of lysozyme. Strom (1955, 486) discovered a large amount of fibrinolytic activator in tears, which probably keeps the lacrimal and Meibomian ducts free from clotted fibrin. Radnót and Németh (1956, 385) found that testosterone administered to female and castrated male rabbits caused the lacrimal glands to increase their weight until 2 or 3 times. It is sometimes seen in malnourished children, that the tear secretion is lessened (Oomen) and it seems probable that the epithelial changes found in the lacrimal gland in vitamin A-deficiency have an influence on the composition of the tear fluid.

b. Meibomian secretion. Kurose (1930, 219) attributed the foam seen at the outher canthus to hypersecretion of the meibomian glands. Eppenstein (1932, 106) also felt that foam is formed by hypersecretion of the fatty meibomian product, but Krückmann in the discussion thought that it was not a hypersecretion of the glandular product itself, but of epithelial debris from the ducts, because the foam is stable, which points to protein beside the fat, as a component. Asahina (1933, 9) found the product of meibomian glands to be olein and palmitine formed in the fundus of the glands, whereas in the neck of the glands cholesterin and glycerin were formed. On the other hand did Linton et al. (1961, 241) not find any free fatty acids or cholesterol, but various other lipids. Norn (1963, 326) found the foam at the outer canthus, as seen in large amounts in conjunctivitis, exophthalmos, painful intrabulbar affections, to consist of fat and fat-imbibed epithelial scales, i.e. products of the lid margin;

according to him foam formation is promoted by blinking (which is thought fanciful by McLaren 256, p. 175) whereas it is reduced in lagophthalmos, and normal in conjunctivitis by infectious causes and keratitis sicca.

It seems probable, that by the keratinizing process which in Xerophthalmia extends to the meibomian ducts, their secretion and desquamation will have an altered composition.

c. Mucus cells in the conjunctiva. In the conjunctiva are found goblet cells, monocellular glands producing mucus, which are essential for keeping the eye moist and smooth. They are most dense in the lower fornix and on the caruncle, less so in the upper fornix ; in the bulbar conjunctiva they are more frequent nasally than temporally, and can altogether fail in the temporal exposed part of the conjunctiva bulbi (Wolff, 579). Kessing, 1966, 200).

DIAGNOSIS OF XEROPHTHALMIA.

In recent years the picture of Xerophthalmia has been very clearly described by McLaren, Oomen and Escapini (257), with beautiful photographs. It would be useful if all doctors in endemic areas could get a copy of this article from the WHO Bulletin.

Xerophthalmia can be divided into 3 stages :

I. Conjunctival xerosis, drying of the conjunctiva, with or without Bitot's spots.

II. Corneal xerosis, reversible changes of the corneal epithelium, which also becomes dry, later showing grey patches.

III. Irreversible corneal changes, beginning in deep epithelial layers, then involving the corneal stroma, leading on to loss of substance, perforation, possibly keratomalacia. Depending on the size of the lesion blindness or serious impairment of vision will ensue.

In many cases the conjunctival signs will be preceded by nightblindness. Essential for the diagnosis is that the changes will react favorably to treatment with vitamin A. Tests for vitamin A-levels in serum will in most cases be too complicated in daily practice in endemic areas, and as there is no risk in treatment with vitamin A in reasonable dosage (up to ca. 200.000 IU in the first week) this is the best and fastest method to clinch the diagnosis. Postponing treatment until the diagnosis has been "scientifically confirmed" can cause loss of the eyes, as the corneal complications can develop dangerously quickly. Nightblindness often will vanish in 4-5 days, xerosis of the conjunctiva shows distinct improvement inside a week as do lighter corneal changes. The serious corneal complications take longer to heal, but they will start improving usually in 5-8 days if the patients are otherwise correctly taken care of (general diet, treatment of infections, infestations etc.). The presence of bacillus Xerosis, which was thought by Pillat to be a sure diagnostic sign (1930, 367) is not found in all cases (Bloch, 31). Salus (408) thought that only in the presence of these bacilli in a xerotic conjunctiva true Bitot's spots would develop, but this was not so in our experience in Surabaja.

Importance of Bitot's spots for the diagnosis of Xerophthalmia. It is characteristic of these spots that they constitute an area of abnormal keratinized conjunctiva on which a white layer will form, that can be wiped off, but will reform after a while. The spots can be (classically) triangular, base against the limbus, mostly in the temporal exposed part of the conjunctiva bulbi, but not so rarely on the nasal side also. Frequently also curved lines are seen, parallel to the limbus temporally, like ripples of wet sand on the beach. The white layer can be foamy, or more solid looking, in the latter case sometimes not pure white but more yellowish. The foamy substance can be so excessive, that it is pushed on to the cornea, or is found floating in loose particles in the conjunctival sac, and on the lid margin, The spots can be easily discerned as soon as they have reached certain size, as the background conjunctiva mostly is slightly pigmented providing contrast. Diffuse conjunctival pigmentation is not normally seen in young Indonesian children, although local pigment nevi are more frequent than in the white race, but it is a sign that something is wrong with the eye, e.g. vernal catarrh, Xerophthalmia, and formaly also Trachoma treated with silvernitrate. Oomen writes that a slight pigmentation is noımal in Indonesian children, but that is not my experience. The pigmentation of Xerophthalmia disappears slowly but completely after healing, not leaving any trace at the site of Bitot's spots either.

Bitot's spots are also to be found, although rarely in Indonesia, in cases where no other sign of Xerophthalmia can be elicited, and where serum vitamin A-levels are normal. Sie Boen Lian described a series of these cases, 18 men, one woman, no children (GTvNI, 1938: 665-671). Métivier (AJO **24**: 1029, 1941) saw Bitot's spots on Trinidad, refractory to vitamin A-therapy. Mc Laren and Paton described probably the same from Addis Abbeba (254) in schoolchildren. From other regions there are reports on single or very rare cases. Paton (357) recently again stressed the non-specificity of the spots. Gödüren and Örgen (1958, 146) saw Bitot's spots in 2 Turkish women without vitamin A-deficiency; no recurrence after excision. Bietti (KMA **143**: 321, 1963) describes a syndrome with xerosis conjunctivae and iris malformations, which he saw for the first time in 1943 (H. J. Meyer, KMA **147**/1: 26-31, 1965).

Bitot's spots in exposed parts of the conjunctiva with chronic irritation probably also has nothing to do with vitamin A-deficiency (Maume nee, 278) but possibly they can be a pointer to an explanation of the formation of the typical spots. These are probably caused by an interplay of several factors :

1. The conjunctiva in the typical "Bitot" area is normally poor in goblet cells which in Xerosis completely disappear ; 2. the lacrimal as well as the Meibomian glands take part in the xerotic process. This probably causes changes in their secretory product, and in the debris shed by them ; 3. the conjunctiva is thickened, folds will be higher than normal, especially in the xerotic area ; 4. slight photophobia and feeling of dryness will probably induce the patient to blink more often than usual, or the normal blinking combined with the unusual composition of tears and Meibomian secretory products is enough to produce foam ; 5. this foam will stick especially to parts of the surface which are rougher than usual.

All of these factors, except the second one, could equally well be operative in eyes chronically exposed to strong light, heat, dry air, dust and other irritants, and thus cause Bitot's spots not related to vitamin A-deficiency. High content of Ultraviolet in the light could be an extra irritant which possibly could be the case in Addis Abbeba because of the height of its location.

Conclusion : While admittedly there exist Bitot's spots not caused by vitamin A-deficiency, at least for East Java I am of the opinion that Bitot's spots in children under the age of 10 ARE caused by vitamin A-deficiency and should be treated as such. Bitot's spots by exposure only are a rarity in this area and only seen in adults. Because of the humidity and almost permanent greenness and the relative lack of dust, there are not as many irritants in this part of Java as there seem to be elsewhere, so the "exposure syndrome" is not frequent at all. But it is on the other hand quite certain that the general diet, and even more that of young children, is poor in A -sources (and in fat and protein) making the chance for development of Bitot's spots of Xerophthalmia all the higher.

Diagnosis of corneal lesions of Xerophthalmia. In connection with the conjunctival signs of dryness and diffuse pigmentation, which are usually present in slight corneal xerosis, the diagnosis in an endemic area will usually not present didfficulties. The first sign is a dryness of the cornea, especially in its lower half, especially on forcefully keeping the eye open for 10-20 seconds (Pillat's sign, 366). Next there develop grey patches in the cornea, deep in the epithelium, gradually involving the stroma also. In this stage not so very rarely a hypopyon will be seen, and if the process goes on without treatment, it will be followed by loss of substance of the cornea, perforation, appearance of an iris knuckle, expulsion of lens and vitreous, and finally phthisis bulbi. The whole sequence can stay free of signs of secondary infection, but if this intervenes the eye will probably be destroyed by panophthalmia. Treatment, even if there is already a hypopyon, can still save the eye, but after loss of substance of the corneal parenhym their will remain scars, and after perforation of the cornea leucoma adhaerens will ensue. This tends to become somewhat smaller in months or years, as the young cornea has a great power of regeneration, but it can also happen, that the iris pigment, which is in contact with the back of the cornea will spread over it in a brown film around the site of the synechia anterior, causing slow deterioration of visual acuity. It is therefore not possible to assess accurately the amount of visual damage caused by xerophthalmia in a recent case, as it can as well improve with time as deteriorate. Leucoma adhaerens can also cause secondary glaucoma, which will need surgery.

Often the corneal lesions are symmetrical (in ca. 75% of cases) which will make the diagnosis easier, as this almost only is seen in Xerophthalmia. There is a distinct preference for corneal lesions in Xerophthalmia to develop in the inferior part of the cornea, although there are exceptions. By these signs it is often after years still possible to make a tentative diagnosis of healed Xerophthalmia, which can be affirmed if the histrory says that the lesion developed in early childhood, between 1 and 5 years of age.

Malnutritional keratoconjunctivitis. Blumenthal (1950 and 1954, 33, 34) described a type of corneal disease which he saw in malnourished subjects, and which showed corneal softening from inside out, with perforation and healing with leucoma adhaerens or anterior staphyloma. He never saw xerosis in these cases and feels therefore that this morbid picture cannot belong to the vitamin A-deficiency syndrome, especially as vitamin A therapeutically is of no value, as are other vitamins. Until now it has been only found in Bantu negroes in Africa, and according to McLaren who saw cases himself, the characteristic sign is local softening of the cornea with a "clean prolapse" of the iris. He calls it "discrete colliquative keratopathy" and saw it often in seasonal accumulations, only in malnourished pre-school children. Strangely the sequelae in later life among the population are not seen : either the disease is a "new" one to the region, or the patients die early for some as yet unknown reason. The only parallel with Xerophthalmia seems to be that the foci of corneal softening also prefer the lower half of the cornea.

Causal factors associated with Xerophthalmia. In cases of nightblindness or slight conjunctival xerosis the patients make an otherwise healthy impression but in more serious cases it is usual that other signs of nutritional deficiency can be found True Keratomalacia is almost never found in children who are not seriously ill.

Liver disease. Purtscher (1900, 384) gave a description of nightblindness, conjunctival and corneal xerosis, keratomalacia and also of retinal lesions, in cases of liver disease (ophthalmia hepatitica Baas). Wright (cited by Feigenbaum, 114) in 1931 saw frequent liver disturbance in India combined with Xerophthalmia. Wagner (1925, 543) describes children who probably because of liver damage developed Xerophthalmia on a fatfree diet given against it. Houet and Weekers (1949, 181) report on twins, one of whom had icterus gravis and keratomalacia, whereas the other twin was healthy. Another child suffered from hepatic disease and Xerophthalmia, and was cured by vitamin A injections. M. Straub and Schaberg (1950, 484) had the opportunity to compare the pathological findings in victims of the hungerwinter 1945 in Rotterdam with malignant malnutrition as found in beggars at Djakarta in 1946. They found in the Rotterdam victims a liver atrophy, whereas in the Djakarta patients, who had always lived on a much lower vitamin A intake, a very low vitamin A-content of the liver and more often than in the Dutch a liver cirrhosis (which they attribute to the combination of chronic vitamin A-deficiency and infection) was found. Lie Kian Joe and Soetomo (1954, 236) in children in Djakarta often found hepatic fibrosis, with and without malnutrition. In serious malnutritrion, where children could be examined repeatedly by liver biopsy it seemed that the fibrosis did not increase with time. If it happens, it must be a very slow process. Fatty infiltration of the liver even in serious cases of malnutrition heals completely. W. Straub and Schmidt (1966, 485) examined dark adaptation in liver patients and found a slower and incomplete dark adaptation with all available methods, including electroretinography.

That liver disease can be a factor in the causation of nightblindness and Xerophthalmia is understandable, as the liver is the most important storage place of vitamin A(beside possibly subcutaneous fat) although it seems that pathologically the signs in children are not impressive. Pillat (BJO,1929) found adult cases with normal liver functions in Xerophthalmia.

Intestinal disease. Already in the descriptions of older "epidemics" the frequent occurrence of intestinal disease prior to Xerophthalmia is mentioned (Mori, cited by Wille, GTvNI 1919,p.425) from Japan; Blegvad from Denmark, epidemic of 1915-1919,cited by v. Szily, 496 ; Birnbacher, in epidemic Vienna, 1919-1924, 26; adults in China, Pillat, 365). From tropical countries there are many reports also : Hoogenkamp in Borneo, Indonesia ("Ontwikkeling, voeding an voedingstoestand van zuigelingen en kleuters by de Ngadju-Dajak op Kalimantan-selatan, Zuid-Borneo, Indonesia" Thesis Amsterdam, 1956), Nguyen Dinh Cat (1958, 322) from North Vietnam. Oomen, McLaren and almost all other authors on the subject stress the importance of intestinal disease previous to the outbreak of Xerophthalmia, although there are some exceptions. It seems reasonable to attribute Xerophthalmia which flares up after intestinal disease to impaired resorption of the vitamin-A-sources from the food. If by a general low supply in the diet of these foodstuffs the children have not had an opportunity to build up good liver stores of the vitamin, the stage where first nightblindness and ensuing Xerophthalmia emerge, will soon be reached.

Intestinal helminthic infestation. Infestation especially with Ascaris is often accused of being a causal factor in Xerophthalmia. Tyssen even claimed that he never saw Xerophthalmia without Ascaris. There seems to be a slight discrepancy in that resorption of vitamin A-sources takes place in the upper intestine, whereas Ascaris mostly lives in the lower part. But we have seen cases where Ascaris appeared practically in all cavities in a child, being coughed up, vomited, creeping from mouth and nose, so the argument of Bose pointing out the local discrepancy ("Nutritional factors in the causation of blindness" 1962) does not always seem valid. On the other hand it is claimed by Tiwary (1962, 512) that Xerophthalmia can be healed much quicker if helminthic infestation is treated (especially hookworm, in his experience).

It is possible that there exists a link in the supply of vitamin A sources and chances for infestation : Tadasu Saiki (1937, 497) found in animals that vitamin A reduced the chances for infestation with Taenia.

The child population in Indonesia is generally heavily infested with various kinds of helminths making it uncertain if there is a causal link with Xerophthalmia or only an accidental one. Hsiao Liang Lin (1937, 239) states that betelnut and garlic can be used as anthelminthics.

Infectious diseases. Many authors call attention to the fact, that previously to the oubreak of Xerophthalmia the patient often has suffered from an infectious disease. In endemic areas the combination with Tuberculosis is not rare at all, but mostly acute infectious diseases are triggering the outbreak of Xerophthalmia in children who live on an indequate diet. In certain parts of Africa (Wessels and Johnson, 1957, 560, also mentioned by Thygeson, 511) Measles seems to be an important cause of blindness; in Indonesia also mumps, otitis media, pyodermia, varicellae can be the trigger. For overcoming an infectious disease with fever a heavy toll is taken of the vitamin A-stores in the liver : as these usually are already small in endemic areas, a general outbreak of Xerophthalmia can follow an epidemic of a children's infectious disease. Nguyen Dinh Cat (1958, 322) attributes the winter peak in Xerophthalmia in that country (North Vietnam) to respiratory infections. In his series of Xerophthalmia patients 30% had Measles, 25% diarrhoeal disease, and only 2% had intestinal worms.

Malnutrition. In many tropical countries protein-calorie malnutrition is rampant, and in certain regions it is often accompanied by Xerophthalmia, often going on to Keratomalacia. This is especially the case in areas, where the usual diet contains few vitamin A-sources, whereas in other parts of the world it is not mentioned as accompanying malnutrition, e.g. Venezuela (Tovar-Escobar, 1955,515,) Sudan (Mamoun yousouf 1967, 261) and parts of Africa where red palm oil is the usual cooking oil. In the original description of "Kwashiorkor" by Cicely Williams eye signs are not mentioned. The word means (Went, 1955,558) disease of the deposed child, referring to the situation where a child is weaned because the next one is born already. These children are in danger of incurring malnutrition as the attention of the mother is centred on the youngest baby, and the weanling child is not yet able to look after its own needs. Voorhoeve (1966,548) found Xerophthalmia in Ibadan, Nigeria, (where it was not known to occur to the pediatricians, although the oculist knew it).

As the connection between malnutrition and child mortality has been known for a long time, there has been done much work on nutrition and malnutrition, surveying population groups, evaluating hospital records etc. : Straub on Sumatra (1927,483) de Haas et al. at Djakarta between 1935 and 1940, Bosma at Medan, Sumatra (1941, cited by Su'dan (488), Sajono in postwar time at Djakarta (Paediatr. Indon. 4/4: 236-247,1964), Su'dan at Makasar (1967,488). For Djakarta the range of infant mortality ranges from 58-156 promille over 1957-1961, depending on the socio-economic condition of the urban area, for Makasar in 1965 it was : 128 promille. Infection and malnutrition are the usual causes of high infant mortality (de Haas et al. 1940,162, Poey Seng Hin, 1957,376 at Djakarta, Timmer: ("Child mortality and population pressure in the DI Djogjakarta, Indonesia "Thesis, Amsterdam, 1961). For Indonesia it can be stated that almost always serious Xerophthalmia is accompanied by serious malnutrition, and the other way round (van Stockum, 1938,482, and the authors already enumerated). Nutritional surveys which have been done in Indonesia therefore have always given much attention to the supply of vitamin A sources beside the usual nutrients. The large prewar surveys of the Institute for nutrition collected valuable material and conclusions (Koetowinangoen Report, 1934,328, Segalaherang-Patjet Report 1937,350, Rengasdjengklok Report, 1940,393). Generally it was found that the usual food of the people in many areas on Java contained : little animal protein, little fat and in regions where rice was plentiful also little vitamin A-sources. If rice was not the staple-food but maize or cassave, the supply with vitamin A-sources was somewhat better, as they contain more carotenoids, and usually are supplemented with more greenstuff. The paradox emerged, that Xerophthalmia was found in the well-to-do region of Patjet, where especially small boys got very little elso to eat but rice, and the lack of Xerophthalmia in South Jogja, where the usual food shortage forces the people to eat whatever can be found, i,e. green leaves of any edible kind. (Timmer, l.c., Bailey, 1961/62,12). Lauw Tjin Giok et al. (1962,226) compared the nutritional status in different socio-economic levels of 2 villages in west Java; generally it was sufficient, but in vitamin A-sources the poorer families were worse off than the others. Signs of Xerophthalmia were only found in the poorer families.

Blankhart at Bogor, West Java (1967,30) studied the individual intake of food in young children. Whereas the vitamin A-sources intake of the family as a whole was sufficient, young children of under 5 years got relatively little of it, but they got satisfactory portions of other nutrients. If the mothers were used to eat plenty of carotenoids, their children tended to have less signs of xerophthalmia. Two reasons could be given for this : the children had been able to build up larger liverstores of the vitamin already during intra-uterine life, and possibly mothers who ate greenstuffs woud sooner give them to their children. Of the whole group (workers in the lower ranks at the university) the family diet was marginal, which makes the most victims in the children of the 1-5 yr. age group, who are growing fast and usually have intestinal worms and suffer most from infectious diseases.

In these circumstances only a little extra like a cold, or measles is sufficient to trigger the outbreak of Xerophthalmia.

In neighboring countries Stahlie in Thailand (1957? 477) found signs of Xerophthalmia especially in schoolchildren, and nightblindness in pregnant women, which vanished after delivery. Couvee et al. (1962, 65) found in the Central Highlands of New Guinea (now Irian Barat) dry corneae among other signs of malnutrition, whereas the oculist Schubert (1963,420) found in various parts of the islands active and healed cases of Xerophthalmia, depending on the local food supply. Willyarto Wibisono, the doctor who accompanied the expedition to the Sukarno-top (1965,577) found that part of the vegetable A-sources were not (yet?) used by the population as food.

Sharma in Burma (1955,425) describes many of the popular habits and prejudices among the population which obstruct a rational nutrition, which in that region especially causes infantile beriberi among other forms of malnutrition. Wolff (1965,580) writes in an interesting article that almost wholly could apply to Indonesia on "the Meanings of food" in Malaya, that although Malaya compared to other regions is rather well-to-do, the popular diet is monotonous, and that especially young children after weaning by a complete lack of regular eating habits of the family and by lack of maternal supervision of what the child eats and where, tend to get far too little of the available vitamin A-sources.

For the assessment of nutritional status in a population Jelliffe has written a comprehensive guide, which in the case of doubtful signs of vitamin A-deficiency (i.e. Bitot's spots) recommends a small-scale double-blind therapeutical trial with vitamin A-therapy. Other signs like corneal Xerosis and Keratomalacia he seems to find sufficiently well-etablished as sure signs of vitamin A-deficiency (1966, WHO, 189).

Pregnancy. In the European context (Vienna epidemic after World War I, 26) attention has been given to nightblindness in pregnancy, which healed after delivery. Wright (cited by Feigenbaum 114) and A. Fuchs (1936,130) saw that in Mysore, India, nightblindness and even Keratomalacia was occurring in lactating women, whereas their children did not show Xerophthalmia. In Surabaja we saw a very few cases also. Mothers with Xerophthalmia nursing children with Xerophthalmia have been described more often (Wille, "Een leerzaam geval van hemeralopia idiopathica cum Xerophtalmia", GTvNI 1919.420-439). From Schiele (416) on there have been trials to give the nursing mother codliver oil, not the child. This would only be justified if the child'sXerophthalmia has not progressed too far.

Co-incidence of other deficiencies with Xerophthalmia. Weech (1930, 552) reported on children who at the same time with keratomalacia suffered from : rickets, from beriberi, from scorbut and from "edematous disease". Liem Tjay Tie at Djakarta (1936,237) saw twins, $3^1/_2$ months of age, whose mother had died, father positive Wassermann test, where both children had Keratomalacia, and one also rickets. Posthuma (1938, 380) also at Djakarta saw a 2 year old orphan who had at the same time Keratomalacia, beriberi and pneumonia. In Indonesia rickets is rather rara and it is interesting that this child never was brought out-of-doors. Maasland (1941,251) reported from the island Nias, where the staplefood is cassave and coconut products, that Xerophthalmia is almost absent, as yellow cassave is consumed, whereas rickets is to be encountered.

Relations between malnutrition and endocrine glands. Wright (1931, cited by Feigenbaum) in south India drew attention to possible affections of the adrenals in Xerophthalmia. Schaberg and Straub (1953,413) saw atrophy of the adrenals in hunger victims, and emphasize that chronic malnutrition is a permanent stress in the sense of Selye. The condition of chronic stress impedes the alarm reaction to renewed acute stress, like infections etc. The normal excretion of 17 — ketosteroids in urine in Indonesians is rather low compared to figures from Europe, but not compared to those from other Asian couttries (Soedarminto and Asri Rasad, 1962,454).

Hartz on Curacao (1949,168) found pancreatic atrophy in malnutrition, in which the acini were involved, but the islets remained intact. From Africa this was also reported, and Straub (152,484a) remarked in a discussion with Trowell on this subject, that the same type of pancreas lesions which in Uganda had been seen in Kwashiorkor were encountered in the Rotterdam hunger patients in 1945. Davies (1952,77) also states that atrophy of the pancreas and sometimes also of the lacrimal and salivary gland is found in Kwashiorkor. Ullerich and Witte (1962,527) discribe a 6 months old baby, suffering from cystic fibrosis of the pancreas, who had also Keratomalacia. Pruys (1925,382) reported on the combination of malnutrition and thyroid ailments as seen in Mid Java. Wendt (1935,557) found a variable carotene level in the blood serum of patients with myxoedema and cretinism, although it was already known clinically as well as experimentally in animals, that vitamin A and thyroid hormone in some ways act antagonistically. Horvat and Maver (1958,178) treated on the island of Krk in the northern Adriatic the school children with vitamin A, because the local food contained very little vitamin A — sources, and found improvement after 3 months in 44%. Pearlman and Burian (1964,359) examined patients with thyroid dysfunction electroretinographically, and found some changes : the amplitude of the be-wave (Scotopic vision) was larger in thyreotoxicosis, becoming lower on therapy, whereas in hypothyreoidism the reverse was found : small amplitude, getting higher on treatment. Whether this points to a retinal factor (via rhodopsin and vitamin A,) or a heightened neural excitability caused by thyroid hormone could not yet be determined.

Still unsolved is the question, whether **bladderstones**, which are quite frequent in South East Asia in young boys, are related to vitamin A-deficiency or not. De Langen (1929,222) reported that experimental animals with longstanding vitamin A-deficiency slhowed the formation first of bladder-later on of kidney-and gallstones, as had been stated by Yoshimoto Fujimaki before. In the rural hospital Modjowarno between 1910 and 1928 70% of the many patients with bladderstone were under 15 years old. Sampoerno (1928,409) published a paper on a small child with Xerophthalmia in which post mortem a kidney stone was found. Reddingius (1938,390) saw very many bladderstones in boys under ten years of age at Djakarta, whereas kidney stones occurred at an older age. Bladderstone formerly was quite frequent in Europe, but has almost vanished. Stahlie (477) found endemic areas of bladderstone in children in Thailand. In the same country Aree Valyasevi and S.B. Halstead ("Epidemiology of bladderstone in Thailand" 2nd. Afro — As. pediatr. congr. 1964) did an extensive survey and found a high prevalence in rural, not in urban, areas, mostly in children under 5, and, what seems important in connection with a nutritional causation, recurrences in 8% after surgery; persons sharing the same food and household surroundings with a bladderstone patient had twice as much chance of developing stones than the average population. Oomen (1936,341) saw Xerophthalmia in North Celebes, but few bladderstones, and de Haas in the discussion on Reddingius' paper commented on the rarity of the combination of Xerophthalmia and bladderstone in the same patient, although epidemiologically both have much in common.

Socio-economic factors. In severe Xerophthalmia like in malnutrition there is often a distinct influence of socio-economic factors, e.g. poverty, low hygienic standards, food taboos, prejudices, death or illness of the mother, mothers working in factories or on estates etc. Depending on the region these factors can be present in varying combinations. Contrary to the usual situation in general malnutrition, in Xerophthalmia it can happen that poverty is not present: in the Indonesian/Malayan context even among well-to-do families the idea is prevalent, that rice or even better: white rice, is by far the most superior foodstuff, the only one worthy to be given to one's children. Most other kinds of foodstuff are regarded as inferior, so that by sheer ignorance of a growing child's real needs, it can happen that Xerophtalmia is more frequent in relatively prosperous regions than in poorer ones. In areas verging on famine Xerophthalmia tends to be rare, as by necessity everything edible is consumed, including green foodstuffs, which ordinarily would be avoided.

The importance of the composition of the family, whether the child lives long enough under the care of its own mother, or is given away to some childless woman, a sister or a neighbor, has been analyzed in some surveys on malnutrition, i.a. by Brown and Opio (1966,44) in Uganda, where of 197 children admitted to hospital because of malnutrition, 60% lived with both parents, 12% with the father alone, 6% with the mother alone.

In our study of Xerophthalmia we tried to investigate the socio-economic factors also, in a number of cases.

Rachma F. Boedjang (1967,36) studied mental development of children after malnutrition, on which in the literature there are conflicting opinions. Most probably the moment at which malnutrition strikes is important, i.e. whether before or after essential periods of development are fulfilled.

Estimation of vitamin A-sources in foodstuffs, serum and milk.

In Indonesia interest in vitamin A and its chemical estimation emerged in the later twenties and thirties: in a series of investigations by Spruyt (1934,475), van Veen (1937,532), van Veen, Lanzing and Agoes (1937,533) methods for the estimation were worked out and applied. The latter found in healthy prisoners on a vegetarian diet serum levels of vitamin A of 8 IU/10cc and carotenoids 8.6 gamma/10cc serum. This vitamin A-level was almost the same as that found in Europeans, but for alpha-and betha-carotene the values were much lower than in Europeans. They thought this was caused by the total conversion of carotenes into vitamin A which itself was not supplied in the diet, whereas the European diet provides vitamin A itself, leaving more convertible carotenoids in the bloodstream. Generally in these studies there was no strict parallelism between vitamin A/carotinoid level in the serum and clinical signs of xerophthalmia in individual cases. Sie Boen Lian (1937,438) published fugures on the vitamin A serum level in Xerophthalmia patients, but his method of calculation was not approved by the biochemists.

Carotenoids in foodstuffs. Lanzing and van Veen (1937) found in addition to previous work by Jansen and Donath, that the best provitamin A-sources are : a certain species of banana (pisang radja bulu): 1000-2500 IU/100 gr.

papaya : 700-2600 IU/100 gr.
yellow and red cassave : 4000-13,000 IU/100 gr.
red palm oil : 76,000 IU/100 gr.

Of special interest has been the estimation of vitamin A-levels in blood of pregnant and lactating mothers, and of their milk. These values were rather low, more so in native Indonesian than in Chinese women in Djakarta (Meulemans and de Haas, 1936,281; de Haas and Meulemans, 1938,258). Cows' milk was poorer in A-sources than mothers' milk. Buttermilk was still poorer, whereas lactic acid milk had not lost its vitamin A-value. The usual way of storage of formulas did not cause loss of vitamin A. It was recommended to add cod liver oil to children's formulas. Lie Goan Hong, Oey Kam Nio and Dradjat investigated human milk again (''Major chemical composition of mature human milk from a selected group of Indonesian mothers'', sec. Afro-Asian cong of pediatrics, 1962) and found it still poor in fat, vitamin A and carotenoid whereas protein is satisfactory. These values agree with those found in India. They make it imperative that nutrition for lactating mothers is improved in these respects.

Population surveys. Donath and Gorter (1938,91) reported on the vitamin A-levels among other data as found in the regions of Gersik, Segalaherang and Tjiandjur, different regions on Java, and van Veen and Lanzing did the same for other areas. The serum levels of groups of children were in agreement with clinical signs of Xerophthalmia and nightblindness (dark adaptation measurements performed in the villages by Gorter). For adults no strict parallelism was found. The importance of the division of the food inside the family was found to be the possible explanation why children had low vitamin A-carotenoid levels, and boys even lower ones than girls.

In connection with the supposed importance of infectious disease for the vitamin A-stores in the body, R. Boenjamin (1951,37) investigated vitamin A and carotene levels in lepers, and found them to be low only in lepromatous lepra, whereas there were no indications that the disease was aggravated by low vitamin A supplies.

Harmanses (1961,167) investigated the vitamin A levels of serum of normal children, (average 64 IU, range 41-110), in kwashiorkor without xerophthalmia (average 41, range 0-72 IU) and in Kwashiorkor with Xerophthalmia (average 31 IU, range 0-41).

From the so-called Sheffield experiment (Hume and Krebs, 428) on human volunteers, it is known that for adults with wellfilled liver stores the time before on a vitamin-free diet the first signs of nightblindness will appear, is very long (one year or more). In children liver stores presumably are smaller, and according to the Expert Committee on medical Assessment of nutritional Status of WHO (1963,565) "in infants the liver stores will be exhausted in 3-4 months". It seems that European children are meant, as it often seems that Indonesian children do not take such a long time to become nightblind : their liver stores are probably much lower still because of the chronic undersupply with A-sources, which operates already in intra-uterine life as well as afterwards.

Rodger et al. (1964,400) found low vitamin A levels in patients with Xerophthalmia, but comments that also in healthy Indians the levels varied enormously. McLaren on a WHO assignment in Jordan found low values and many cases of Xerophthalmia in infants and young children ("Joint FAO/WHO Expert Committee on Nutrition 7th report, WHO technical report series, no 377,1967). Of more than 300 randomly selected children under 6 years of age more than half had levels of less than 20 μg/100 cc.

Serum protein levels in Xeropythalmia. Yap Kie Tjong (Jogja, Java) compared serum protein levels in conjunctival xerosis and in keratomalacia, and found lower protein values in the latter. Timmer (l.c.) gives a table on various values (adapted by me) :

healthy :	number	total protein	album.	globul.	A/G ratio	
Europe		6.3-7.6	4.5-5.5	1.8-2.4.	2.3.	
Java, doctors's children	25	7.4	4.3	3.1	1.4	
orphans	33	7.3	3.8	3.5	1	Yap K.T.
poor children	21	6.9	3.4	3.5	1	
Poey Seng Hin, 2 different methods	33	7.45	3.96	3.49	1.1	
	21	7.5	4.25	3.25	1.3	1-4 yrs
Malnutrition :						
Timmer, Jogja	89	5	2.67	2.33	1.14	7/12-7 yr.
Poey Seng Hin, Djakarta	112	5.26	2.09	3.17	0.69	
	43	5.3	1.87	3.43	0.54	
Xerosis conjunctivae	10	6.2	3	3.2	0.9	
Keratomalacia	10	4.3	1.7	2.6	0.7	Yap K.T

According to Went and Gandasoebrata (1955,559) figures for total protein and A/G ratio in Kwashiorkor as reported in the world literature vary between resp. 4-4.8 and 0.37-1.1. Yap's figures in Keratomalacia cases are well within this range and reflect the usual experience that Keratomalacia is seen in children, who are in a bad general condition, at least in Indonesia. Whether it is possible that Keratomalacia clinically develops in children who are except for vitamin A-deficiency in a completely normal nutritional condition is still uncertain.

Xerophthalmia in Indonesia. The physician of J.P. Coen, Jacob Bontius, gave a description of deterioration of vision which was suffered by seafarers on the route to the Moluccas, which was cured by the Natives with liver, or also liver oil, from the shark (1642,40). From the therapy one would suppose that this has been nightblindness, occasioned by im-proper diet and strong light. Ouwehand (1900,351) was the next who mentioned a kind of nightblindness, seen in boys around Lake Toba on Sumatra in certain years, in some cases showing conjunctival xerosis. Westhoff at Bandung (1911,563) gave a good description of Xerophthalmia, the preceding nightblindness, and ensuing Keratomalacia. In his annual reports he mentions between 15-20 cases of conjunctival xerosis, 3-6 cases of Keratomalacia annually (on total outpatients of 2500-3700). Later at Bandung in 1928 and 1929 Hötte does not mention Keratomalacia, but nightblindness and Xerophthalmia were seen in resp. 9 and 26 cases of 6021 total outpatients, and 6 and 43 in 6929 outpatients. Wille at Semarang found comparable figures for 1913-1920 (572), but Sie Boen Lian at Jogja gave a higher figure for xerophthalmia, not for Keratomalacia ("Aanteekeningen uit het jaarverslag van het Ooglydersgasthuis te Djakarta, 1932", GTvNI, 1933: 559-573.) Wille (1933,574) and Sie Boen Lian (1929,433) wrote papers on their experience with the deficiency, giving many references of the findings of others. Sampoerno (1928,409) mentioned that Sjaäf at Padang saw Keratomalacia even in breastfed infants, whereas at Surabaja medical school there were only seen 5 cases in one year. The travelling eye surgeon Tyssen, who worked especially on Sumatra considered Keratomalacia to be the most important cause of blindness in that island, where Trachoma was rather less frequent that on Java, and where conjunctivitis gonorrhoica was rare (1932,1934, 1936,1940: 520, 522, 523, 524). He claims a different behavior in children under 2 years of age, who become ill with Keratomalacia, as many boys as girls, and children over 2 years of age, where twice as many boys as girls suffer from Xerophthalmia, by a constitutional apraxia of the vitamin A-sources in the diet.

A relation of Xerophthalmia with general dystrophy in children was found to exist by Straub (1928,483) and mentioned by Zeeman (cited by Sampoerno), and in the thirties de Haas, first on Sumatra, later at Djakarta, made intensive study of the whole complex of malnutrition and Xerophthalmia (1936,1937,1940: 156,159,160,162). He estimated in 1937 the vitamin A-level in the serum of a child which was never breastfed and which had Xerophthalmia after diarrhoea, probably one of the first of such estimations. Before treatment there was no vitamin A to be detected in the serum, carotene 6 gamma, after treatmentt 12-15- IU vitamin A and 15 gamma carotene.

Oey Djoen Hoat (1936,329), Goelarso Hadikoesoemo (1938,144), Gardjito (1939,137) in surveys in villages in different parts of Mid Java found Xerophthalmia usually in 2-2$^1/_2$% of children under 6 years examined. Oomen from Celebes mentions that xerophthalmia is not rare in the hills, even leading on to blindness by keratomalacia, sometimes caused by the use of sweetened condensed undermilk (340,341). From Sumatra Tyssen claimed that Keratomalacia had a great influence on the number of blind persons found in a region, and van Manen (1938,262) mentions that almost all blind children under the age of 15 years had suffered from keratomalacia, more boys than girls in the age groups over 2 years.

In the postwar years first Oomen with various others made intensive and extensive studies of the problem of Xerophthalmia/ malnutrition, analyzing factors of : food, infections, infestations, secial circumstances (1951,1953, 1954,1955 : 342, 343,344, 345, 346). He added liver biopsies in a number of cases, and designed a "malnutrition index" based on 10 easy to determine points scored according to a simple system. This index was later used by Hoogenkamp on Borneo (l.c.) and Timmer at Jogja (l.c.) and I tried to apply it in my investigations of Xerophthalmia, reported later.

During the Pilot project on Trachoma Oey Khoen Lian (1958,334) drew attention to the important fact, that Xerophthalmia in certain regions was still on the increase, with more than 80% of cases of Xerophthalmia occurring in the age group between 1 and 5 years. Of the total number of outpatients seen in 1958 by his organization (179.549) of all ages, 2.9% had Xerophthalmia in all stages, whereas 0.3% had Keratomalacia. Of all Xerophthalmia cases 97.7% were less than 15 years old. In a survey during the pilot project in 8 villages xerosis conjunctivae was found in 6.5% of all inhabitants, 0.2% had xerosis corneae, Keratomalacia was not encountered. In villages with a higher intake of vitamin A-sources there was a lower incidence of Xerophthalmia (Blankhart), but there was no strict parallelism : incidence varied from 4/1000 inhabitants in the best-fed village to 13/1000 in a village with rather low intake. But two villages with still lower intake of vitamin A-sources had only 10/1000 and 5/1000 incidence of Xerophthalmia. Oey Khoen Lian in 1961 and 1963 (335,339) again emphasized the importance of Xerophthalmia and recommended his system of a network of policlinics specially devised for eye disease.

Soedjono D. Poesponegoro (1959,456) considers protracted breast feeding, as sometimes recommended as of no importance in preventing malnutrition, including Xerophthalmia, but recommends weaning any way at age 12 months, and earlier if breast milk is clearly insufficient.

Timmer (l.c.) reports on Xerophthalmia patients seen in the eye hospital "Dr. Yap" at Jogja, were from 1952 till 1958 2056 children hailing from DI Jogjakarta were seen suffering from Xerophthalmia: although there were more boys than girls among the patients with Xerophthalmia, the percentage among the children blinded by Keratomalacia was the reverse :

Percentage of girls in total number of Xeropthalmia patients compared to percentage of girls in children blind by keratomalacia.

	—3mn	16 mn	—12mn	1 yr	2 yr	3 yr	4 yr	5 yr	6 yr
girls, Xerophth.	49%	46%	44%	39%	41%	40%	43%	43%	40%
girls, blind by keratomalacia	55%	50%	55%	42%	47%	50%	50%	46%	50%

Except in the age group 4 years it seemed that Keratomalacia made more victims among girls than boys.

Xerophthalmia as seen in Indonesian malnutrition.

From an ophthalmologist's point of view the figures given by Timmer (l.c.) are the most informative, as he clearly differentiates between the 3 stages of Xerophthalmia. In 4 years between 1/7. 1954 and 1/7. 1958 454 children with malnutrition were admitted to Bethesda hospital at Jogja, divided by age :

	—½yr	—1yr	1 yr	2 yr	3 yr	4 yr	5 yr	6 yr	total
total number	30	52	158	108	52	30	16	8	454
mean malnutr. index	13	14	15	15	15	15	16	13	15
Total Xerophth.	70%	24%	40%	65%	73%	78%	77%	62%	55%
specified as:									
conj. xerosis	3%	10%	17%	25%	17%	21%	36%	50%	19%
corneal idem	27%	6%	16%	25%	37%	39%	27%	—	22%
corneal destruction	40%	8%	7%	15%	19%	18%	14%	12%	14%
Bitots spots	17%	4%	16%	25%	30%	33%	27%	25%	20%

380 children with malnutrition were admitted to the children's ward of the University hospital at Jogja between 1/5. 1955 and 1/7 1958. Same as previous table:

total number	24	46	126	88	42	32	14	8	380
mean index	16	15	16	17	17	17	18	17	16
Total Xerophth.	46%	33%	44%	53%	57%	75%	83%	55%	54%
specified as:									
conj. xerosis	9%	13%	14%	9%	20%	27%	29%	44%	19%
corneal idem	12%	12%	21%	25%	20%	21%	29%	—	19%
corneal destruction	25%	8%	9%	19%	17%	27%	25%	11%	16%
Bitot's spots	3%	12%	13%	20%	20%	21%	41%	25%	17%

From these tables can be concluded that very young infants if they suffer from general malnutrition run a heavy risk of becoming blind by Keratomalacia, and that the risk between 2 and 5 years is again rather high.

Oomen had already found a parallelism between the degrees of malnutrition and Xerophthalmia (reported in Outline, 348):

Degree of Xerophthalmia	slight	Degree of malnutrition	
		moderate	severe
Xerosis conjunctivae	14	34	14
Bitot's spots	12	25	5
Xerosis corneae simplex	4	15	2
Xerosis corneae perforans	1	8	12
Keratomalacia	0	0	8
Total number of children	14	34	16

(Children less than 6 years old, on Java: positive Mantoux test, positive malaria smear and enlarged spleen excluded).

From prewar figures of van Stockum (1938, 482) and de Haas and Posthuma (1940, 162) can be seen that of children who suffered from malnutrition and Xerophthalmia, a large number went blind and a large number died. In van Stockum's report from Semarang and Bandung less than half of the children survived without going blind, and the same applies to the patients of de Haas. Possibly even those who lived, did not survive for a long time, especially those who were blinded and thus clearly had sustained serious damage to their metabolism. The theory of Tyssen, that there are more blind men than women, because boys oftener suffer from Xerophthalmia, is very difficult to prove, precisely because nothing is known about the later survival rate of the blind children. From Timmer's figures it would seem that on the contrary more GIRLS are blinded, but it would be possible that 1. the damage done lessened their chances for survival, and 2. that they were not taken care of from prejudice, either from preference for boys generally, or from unconscious loss of interest in BLIND children.

The difficulty in proving these suppositions is evident.

Age and Sex in Xerophthalmia. In all reports published on Xerophthalmia in Indonesia the age group with the highest frequency is from 1—5 years, with a somewhat lower age for the peak in Keratomalacia (around $1^1/_2$ year) and a somewhat higher age for the peak in Xerophthalmia generally (around 2—3 year).

In Indonesia also it can be said to be probably true that boys are attacked more often than girls, (Sie Boen Lian "Zyn er inderdaad meer mannen dan vrouwen blind door Xerophthalmia?" GTvNI 1937 : 3283—3286) ; van Manen, 1938, 262 ; Gomperts, 1940, 151 ; Teng, 1963, 504 ; Tamin, 1966, 498).

Mc Laren in a recent survey in Jordan did not find any difference in sex (1.c). Apart from social customs it could be that only in certain regions the boys get less from the available vitamin A-sources, and that they are more exposed to strong sunlight. In a recent small survey in part of the town of Surabaja (Hoepoediono, as yet unpublished) again, just like in the greater prewar surveys, the number of boys attacked by Xerophthalmia was greater than that of the girls. Of figures collected in outpatients' departments it would be possible that they were biased, if really parents would bring their sons sooner than their daughters, but population surveys should be more trustworthy. It seems that the percentages found in the small hous-to-house survey agree closely with the figures found in the Undaan outpatients' department, except that in the former there are no serious cases of Keratomalacia, which were already under treatment in one of the hospitals. The incidence with age was also very similar with other results in Indonesia, and also with other figures from Surabaja (Tamin, 498).

Elliot (104) and Bose (1.c.) from India also say that boys are more often seen suffering from Xerophthalmia.

Seasonal peak. From many countries there are reports on a seasonal peak in the incidence of Xerophthalmia. Over a hundred years ago the "epidemic" at the end of the fast before Easter was already known in Russia. Mori (cited by Wille, 1.c.) in Japan found a peak from July till September, attributed to the diarrheal season ; Elliot states (104) from Madras, that Xerophthalmia is most frequent in famines, and further in the clear weather season; Bose (1.c.) on the other hand says that in Calcutta and Bengal the incidence is not higher during famines, but in the rainy season, also the diarrheal season ; from Europe, Bloch (1920, 31) remarked on the higher frequency in Denmark in the spring, which he attributes to the increased growth of children in that season ; Toller (1922,

513) at Vienna saw almost all cases in children in May, also in the period of increased growth (from March till August) ; in China (1929) Pillat saw most cases in March-July.

In Indonesia Mulock Houwer was the first to draw attention to a seasonal peak at Djakarta during the dry season, from June till August, Whereas Pranowo saw no such increase at Surabaja at the time (Both in : Hand. 7e. Ned. Ind. Nat. Wet. congr. 1936, p. 197—201.)

Van der Schaar ("Geopsychische en geophysische verschynselen", GtvNI, 1940, p. 706—779) found the growth curve in school children in Mid Java to be slowest from April to August, rising in September till a peak in December-January.

Corcos and Besnianou (1955, 63) in Tunis attributed a seasonal peak at the end of the summer to diarrheal diseases. Salus (1957, 408) in a European concentration camp saw Xerophthalmia in epidemic form early in spring "due to the activating influence of sun radiation".

Hoogenkamp (1956, l.c.) on Borneo saw a seasonal peak arising after that of diarrheal diseases. His figures for 1953 and 1954 :

Month	enteritis	Xerophthalmia	Month	enteritis	Xerophthalmia
Jan.	430	12	July	1100	31
Feb.	510	11	August	820	22
March	515	11	Sept.	900	43
April	560	24	Oct.	880	48
May	690	16	Nov.	620	27
June	970	40	Dec.	—	3

In this region: rainy season from Dec. to April, vegetables plentiful from **February till April. Dry season starts in June/July.**

The highest peak is seen after the peak in enteritis, but it seems to me that there is already a slow rising tendency earlier in the year, from April on. In my own calculations I did not rely on general curves, but investigated the previous diseases in each child individually, and in that way there did not appear to be a parallel to diarrheal disease at the peak season (see later).

Nguyen Dinh Cat (1958, 322) found in North Vietnam two seasonal peaks, one in winter, attributed to respiratory infections, and the other in summer, in connection with enteritis.

Blankhart (1967, 3) found a higher incidence in Xerophthalmia at Bogor, West Java in the months Sept.-Dec. attributing this to higher rice prices, scarcity of vegetables and prevalence of diarrhoea.

Influence of light. Whether light — especially sunlight — as such could be a factor in the causation of a seasonal peak in Xerophthalmia has not yet been analyzed. There are older reports on damage to the retina by strong light: Jess (1920, 190) found ring scotoma, nightblindness, weakening of the blue/yellow color sense in war pilots, and also in eclipse blindness as a residual lesion. The signs would point to damage to the neuro-epithelial cells of the retina, the rods as well as the cones. Zade (1920, 598) saw the ring scotoma also in workers in the telegraph service who had to look at the sky for prolonged periods of time.

Hecht et al. (1948, 171) found in men who worked as lifesavers on the beach in summer a delayed dark adaptation, which even after several hours in the dark still showed higher thresholds than normal, exposure during several days having a cumulative effect. Use of appropriate dark glasses prevented the phenomenon. Dowling considered (95) that in these cases rhodopsin in the retina had been bleached to a degree which could not easily be regenerated.

Only the visible and short infrared rays of sunlight reach te retina, the ultraviolet part is absorbed by cornea and lens, when in excess irritating both conjunctiva and cornea severely, as is seen in snowblindness and ophthalmia electrica. Ultraviolet light is sooner absorbed in the atmosphere especially by dust and moisture if present, whereas visible and infrared light penetrate to the earth in a greater percentage. From investigations by Vry (1932, 540) it is known that even in the coastal plains of Java still more ultraviolet light reaches the earth than is usual in North West Europe. This is probably all the more so in the months May till August, when the atmosphere is clear. The amount of sunshine on Java has been measured by Boerema and Berlage (1939) and Schmidt (''On the distribution of the duration of sunshine on Java'', 1950, Kem. Perhubungan etc. Djaw. Meteorologi dan geofisik, verh. no 40). At Surabaja there is a peak in hours of sunshine per day from June through November, whereas in Bandung this peak is seen from June through August.:
Duration of sunshine expressed as a percentage of the possible time :

Surabaja :	Dec/Febr.	50-60%	Bandung :	50-60%
	March/May	60-70%		60%
	June/Aug.	80-90%		70%
	Sept./Nov.	80-90%		60%

At Surabaja the later months of sunshine show a less clear atmosphere than the first. This agrees with the seasonal peak of Xerophthalmia at Surabaja. Of the two kinds of light it is thinkable that the ultraviolet part would be an irritant for the conjunctiva/cornea, whereas the visible and infrared part would pass to the retina and possibly cause excessive bleaching, which by the lowered blood supply of vitamin A could not be regenerated in sufficient measure. It is often seen that children with Xerophthalmia keep their eyes closed : this could be an instinctive protective means of shutting off relatively excessive light to the retina, as well as against the feeling of discomfort caused by ''dryness'' of conjunctiva/cornea. If there is any truth in this idea, it would explain why small boys are more vulnerable, who more often play out-of-doors, flying kites etc. than girls, as well as the seasonal variations. Of course the factor light can only be operative in cases where by other causes there already exists a vitamin A-deficiency. Contrary to what McLaren says ,that additional factors probably cancel one another out, it could also be that they potentiate one another in a certain season, depending on local conditions. (meteorological data received by kind help of Ir. Moersidi and W.E. Syatauw, of the Meteorological Service at Djakarta).

Malnutrition and Refraction. McLaren (1960, 254, 255) reported on an increase of various kinds of refraction anomalies in a group of children which had survived a famine, as compared to children from a neighboring region who had been free of famine. The ''famine group'' showed more anomalies (mixed astigmatism, strabismus, anisometropia, axial ametropia etc.) than the other group. Sood and Gupta (1967, 468) found in a group of malnourished children between 7 months and 5 years old on an average higher Myopia than in an otherwise comparable group which had been better fed. The myopia tended to lessen in time, but did not become normal.

Treatment of Xerophthalmia. In treating Xerophthalmia right from the start it must be ascertained whether there is Xerophthalmia only, or whether there exists general malnutrition as well. In many cases both will be present and have to be taken care of. This is a fact which is often forgotten by general physicians and maybe even more so by ophthalmologists.

From thousands of years ago it has been know that liver was the best treatment for nightblindness : according to Hirschberg (173) Egyptian papyri, Greeks, Romans, Arabs, Chinese, Indians knew this already. Medieval Europe knew it and judging from Bontius' story Indonesians knew it also 300 years ago.

All the more astonishing that there still occurs Xerophthalmia!

Trantas père (1900) and Trantas fils (1929, 516) recommended liver therapy for nightblindness also if not caused by liver disease. Schiele (1907, 416) treated nightblindness in babies by giving codliver oil to the nursing mothers, and in Denmark, according to Wille (1. c.) the usual therapy was milk. Wille himself also used this in his first years at Semarang, later changed to liver therapy, and eggs, and after he knew that in Japan cod liver oil worked micacles, he tried it also. As vitamin A was discovered it became clear that this was the active substance in all types of treatment, There is one curious excursion in another direction : Fuchs (130) claims that he cured nightblindness by ultraviolet irradiation of the body, without changes in the diet. I found this nowhere confirmed. After the importance of vitamin A had been established that of the carotenoids became clear, and in 1934 Rosedale and Oliveiro (402) from Malaya recommended red palm oil, very rich in carotenoids. Wendt and Schröder (1935, 556) accentuate that some vitamins are one another's antagonists, which makes it necessary to give a balanced diet without excess of one or another of the vitamins in cases of deficiency. Someswara et al. (1953, 467) investigated an outbreak of Xerophthalmia in an Indian village, where not only a low vitamin A supply but also anemia were operative, making it necessary to treat this also.

As cod liver oil however valuable in light cases often is not well tolerated by sick children, there was made a concentrate of the vitamin itself, which can be given by injection. Thomson (1953, 510) from Malaya advocated it, as well as oral red palm oil and skim milk. Rambo (1958, 387) even postulates that the parenteral route is necessary, especially in severe cases. In corneal perforation he performs a conjunctival bridge-operation. Rodger et al. (1963, 1964, 397, 400) emphasizes the need for protein (milk) supplements to improve the absorption of vitamin A/carotenoids.

In Indonesia, as has been stated before, in many regions the usual diet contains little animal protein, little fat and almost no vitamin A as such. Although carotenoids are plentiful, they are often not used in the diet. Milk and cod liver oil have to be imported, which has stimulated efforts to find local sources of vitamin A. Sihombing (1958, 443) reports on the rather disappointingly low content of vitamin A in shark liver oil as compared to cod liver oil. Poey Seng Hin and Harmanses (1959, 377) found that the best and quickest method of Kwashiorkor therapy was cows'milk, then soybean milk. Sutedjo et al. (1962, 493) tried fish flour in Kwashiorkor and found it in certain forms to be acceptable to the children and a good protein source. But from intravenous treatment with essential amino-acids Sutedjo et al. (1962, 494) saw no result at all. Bailey (1963, 13) reports from Nw. Guinea, that children with malnutrition could be satisfactorily fed with solid peanut foods, which are plentiful in the region. Maman Sanouman and Midian Sirait (1964, 260) claimed that the need for vitamin A-sources could be adequately covered by making the red palm oil and fish (shark and tuna) oils available. Terra (1964, 507) recommended the use of various kinds of green leaves, especially from cassave, which have a reasonable protein content and high percentage of carotenoids. There is a drawback to its use as vegetables viz. that it must be cooked well to destroy its HCN. As long as housewives seem to be rather careless in their cooking habits it is probably not yet the time to recommend the general use of these leaves as vegetables. In 1967 Sutedjo et al. (495) compared deodorized fish flour to other foods, and found it better than local soybean milk as a protein source, but not as good as cows' milk or breast milk. Sathe (1955, 411) from Bombay recommended the support of the usual therapy of angular conjunctivitis (Morax Axenfeld) with vitamin A, as this according to him gives far better results than zinc alone.

Conclusion : It cannot be repeated often enough that the treatment of Xerophthalmia should take into account all of its component factors. The vitamin A deficiency should be treated with vitamin A itself (not relying on carotenoids) to start with in injections, 150,000—200,000 IU in the first week, to which can be added vitamin A in an acceptable oral form. The causal additional factors should be treated if they are present : anthelminthics, antibiotics, protein supplements, balanced multivitamin supplements are mostly necessary beside the therapy with vitamin A. Local treatment of the eyes is far less important: local cod liver oil drops in the eyes if further nothing is done is dangerous. "Curing" diarrhoea with tea-diet only is criminal in regions where malnutrition and Xerophthalmia are endemic : nobody knows for how long the mother will continue this "therapy" if the doctor forgets to say that it is only meant for one day! Prescribing 100 gr of meat for a oneyear old child of poor parents is worse than useless. These things happen, unfortunately. Treatment of Xerophthalmia is well within the province of the general physician, but they should take time to find the best way of treating each individual case within the limits of economic possibilities. There is no sense in sending all Xerophthalmia cases to the oculist, as he is often LESS competent in prescribing the necessary general treatment than a general physician, although even the oculist will from sheer necessity have to learn to treat the whole child, in an effort to lose no valuable time in referring a child to a general physician or a pediatrician. As the development of severe Keratomalacia is a fulminating process no child should be referred to another doctor at all without having been given first an injection of vitamin A, at least 50,000 I U, impressing on the parents that this is not enough, but that they should seek further help instantly. McLaren stresses the point that for therapy vitamin A itself is strictly necessary, reliance cannot be placed on red palm oil, although in prevention it is valuable.

In North Mid Java a system of special eye dispensaries has been developed by the late Prof. Oey Khoen Lian of Semarang, which is centred on an Eye Hospital in that city, with facilities for eye surgery. In ca. 35—40 smaller towns and villages around it are eye policlinics, under specially trained eye nurses (male and female). Around the year 1960 they treated over 200.000 patients annually, more recent figures are not to hand. A large part of the children patients suffered from Xerophthalmia : the percentage of Xerophthalmia cases among all patients (adult and children together) rose to 4%. In cases where corneal complications had not yet begun blindness could be prevented by early treatment. This system of specialized eye policlinics in smaller towns and villages brings expert help nearer to the patients, and in that way probably popularizes the habit of seeking early help in eye disease. Especially in regions where the prevalence of Xerophthalmia is high it could be a very useful way of preventing blindness in children. The drawback would be, that possibly the specialized eye nurses would tend to treat only the eyes, not the whole sick child : in Xerophthalmia the additional causal factors should get full attention as well as the general nutrition of the child, which implies education of the parents. In the system it should be guaranteed, that close supervision by the leader-oculist is possible by regular visits to the policlinics, and that the knowledge of the eye nurses would be kept up to date by special meetings of the leader with the nurses for refresher courses etc. As the cost of this type of eye service need not be very high, it would be advisable to determine regionally if there is a need for it. In other regions the re-education of all medical personnel working in the existing servics to impress the importance of all aspects of Xerophthalmia detection and treatment would possibly to be preferred.

Prevention of Xerophthalmia.

Measures of prevention of Xerophthalmia will have to take into account a complex of local circumstances : availability of necessary foodstuffs, customs and habits of the population, age agroups which are at risk, economic factors. In Indonesia attention has been given to the problem in an extensive way, and until now the visible result is that Xerophthalmia is on the increase! Quoting McLaren (Malnutrition and the Eye, p. 217) : "The movement away from the rural areas and the congregation of people in large towns going on at an alarming rate all over Africa and Asia today may be cited as an example of the kind of current trend toward the actual creation of new, rather than the solution of old, nutritional problems". To which can be added, that the rural areas are often not better off in nutrition, because the available sources are not used, and competent help is farther away. To discourage all sort of high expectations for an early and easy solution of the problem another quotation from McLaren (same page): "Generally speaking, however, improvement in nutritional status of populations has only come as the result of a general rise in the standard of living and not because of any specific measures taken to combat disease" This lies far outside the power of ophthalmology, or even of medicine.

Nevertheless efforts are being made to find ways and means to improve the nutritional status of the population. WHO/FAO have studied the requirements for different ages, for pregnant and lactating women etc. (569). The range of the recommended intakes is from 300 μg retinol/day for small children, rising to 750 μg for adults, which also covers the additional need in pregnancy. These figures have to be reduced to quantities of foodstuffs by nutritionists using complicated formulas but it can be simply stated that the best way of improving the diet in areas where as well protein and fat as vitamin A is scarce would be to instigate a movement to increase poultry farming and the better use of greenstuffs which are available. Probably a long period will be needed to accomplish this change, as it probably will have to be taught in school now, to be applied in the next generation. As is well known and again stated by Sharma (425) and Wolff (589) in agricultural communities food habits are very difficult to change, and it can only be done slowly, a step at a time. Veeger (cited by Blankhart, 28) seems to have reached visible results by training Papuan girls as "village advisers" on Nw. Guinea, but probably the local circumstances were rather special in that case.

An easier way out would be the proposal of Prof. Salim (406) to enrich one of the foodstuffs already in general use with vitamin A, a way which FAO/WHO also seems to have in mind (570) but technically this problem has not yet been solved.

One of the sources of carotenoids which is at hand in Indonesia, but not yet sufficiently used, is red palm oil. Trials by Oey Khoen Lian in Mid-Java (339) and in West Java with small doses were successful in preventing Xerophthalmia (Oey Khoen Lian, Liem Thay Tie, C.S. Rose, D. D. Prawiranegara, & P. György: "Red palm oil in the prevention of vitamin A deficiency, A trial on preschool children in Indonesia", Am. J. Clin. Nutr. 20 : 1267, 1967). If red palm oil could be brought on the market in sufficient quantities, maybe mixed with coconut oil which is more in agreement with the popular taste, and if it could be popularized as cooking oil, much would have been won. Most of the red palm oil which is produced is exported to Europe and USA, where it is used in the fabrication of margarine : the carotenoids have to be destroyed first in the fabrication, and afterwards vitamin A has to be added by legal prescription. . It seems simpler if the red palm oil is used for Indonesian children straight away, although this is detrimental to the earning of foreign valuta.

It would be possible also to establish a procedure whereby all babies were given one shot of vitamin A shortly after birth, enough to tide them over the danger period. If this is workable, how large the dosage would have to be, is not yet known. A difficulty would be that whereas children born with expert help could probably be reached in this way, a large number of children is still born with unqualified help, and for this group the preventive measure would be all the more necessary, but they would present difficulties in reaching them in time.

Conclusion : prevention of Xerophthalmia (and of malnutrition generally) is still a very urgent but so far unsolved problem, which nevertheless must be given constant attention.

CHAPTER TWENTY FIVE.

XEROPHTHALMIA as seen at Undaan, Surabaja.

1. Preliminary. Because during the present study it became once again clear, that in spite of the existence of many other important blinding diseases, Xerophthalmia is still the prime cause of blindness in chil-

dren, it was felt to be useful to add a special study on this subject. Blindness in children is from the viewpoint of public health of the greatest importance: apart from the personal grief of the blind child and its family, the social and economic consequences which are involved in a relatively high number of blind children in a given community, are very great. They need extra provisions for their education, afterwards special facilities to give them suitable vocational training and guiding, supplemented by services for job finding. Generally it can be said that preventing blindness is saving public money, but this is all the more true for preventing blindness in children.

2. Definition. In agreement with present usage (Oomen, Outline, 348) I use the term Xerophthalmia for all types of affection, which usually are attributed to deficiency of vitamin A in the body. To facilitate a differentiation into various stages, which have a very different importance, I gave them the following signs:

Xo. Nightblindness, which has begun recently, and which from the history (food, previous illnesses etc.) is most probably caused by vitamin A deficiency.

XI. Conjunctival signs: xerosis conjunctivae, Bitot's spots (described completely in McLaren et al. 257) are present, with or without nightblindness.

X2. Additional slight corneal affection: dryness of the cornea, especially after forceful opening of the eyes, slight keratitis, still reversible and not leading to serious permanent impairment of visual acuity.

X3. Severe, irreversible corneal affection, until total destruction of cornea, with ensuing serious impairment of vision. Keratomalacia.

X4. Recently healed Xerophthalmia, with corneal scarring to a greater or smaller extent.

Comment: I introduced the signs Xo and X4 to be able to record all cases which had something to do with Xerophthalmia. Usually Xo is either not recorded, or it is brought under X1. X4 is usually not recorded at all, but I needed the sign, to include it in the addition of cases involved in the seasonal variation of frequency. I included only those cases of X4 (presumed healed Xerophthalmia) if from the history it appeared that the eye disease had not been longer ago than 3 months. I then placed the case in the appropriate month.

3. Description of former experience with Xerophthalmia.

From the following tables it can be seen, how very frequent the disease has been through the years. Our figures on Xerophthalmia start before the inception of the present study, in 1955, and are carried on for 10 consecutive years, giving interesting sidelights on other events which took place during this time. We feel that socio-economic factors are reflected in the frequency of Xerophthalmia in an area: after ca. 1957 the economic situation deteriorated, but during the same period centres for MCH were opened in many towns and villages all over East Java, which possibly has had some influence on the height of the seasonal peak, which is slightly less in evidence in 1962. In the following years UNICEF help (skim milk and vitamin supplies) was suspended, which tends to have an adverse influence on the interest of the toddlers' mothers as well as on the midwife-leaders of the MCH. At the same time the general economic situation made it impossible for the public at large to buy milk products on the market. The number of MCH centres in East Java increased between 1953 and 1963 from 151 to 758, certainly impressive figures. (R. Suhasim and Achmad Dipodilogo, Paediatr. Indon. 4/4 : 266-275, 1964).

As far as the Eye hospital Undaan itself was concerned, an important influence could be attributed to the help which the social pediatrician, R.A. dr. Gondosoebarijo, gave in weekly examinations of all the children, who suffered from eye diseases caused by general ailments (Xerophthalmia, Scrophulosis, congenital malformations etc.). It was possible to supplement advice with extra food and medicaments, gifts from various sources, which probably is the reason that many more children were brought to the outpatients' department of Undaan than in previous years. As dr. Gondosoebarijo is still carrying on this work without any remuneration, we have very good reason the be grateful to her for her help. From 1960 till 1966 we could improve on the service given, because we received permission to admit sick children to the children's ward of the Research Institute for Public Health where there was an opportunity to run several tests, which could not be performed in the Eye hospital, and where nursing of the often seriously ill children was more adequate. The Institute at the time was under the leadership of dr. R. Wasito, to whom we are always very grateful for the permission whereas the children's ward was under the supervision of dr. Gondosoebarijo. For financial reasons this service had to be suspended in 1966.

The following table 15 gives the figures for ten years Xerophthalmia, by month, stating the total number of children under 16 years of age, the number of Xerophthalmia cases, also in percentages, and the total number of adult patients and the total number of children patients seen during the year. From 1959 through 1963 the figures for January do not include the leftover cases from the previous December, as is done for administrative reasons in the other years. In these latter years the figures are artificially high, as many cases which are not recent any more are carried over into the new year. This could not be avoided.

4. Xerophthalmia during the period of the present study. 1959-1962.

1959. Total outpatients : 14832 adults/3721 children : 20% children.
Blind children : 185 **boys** under 16 years of age with Xerophthalmia : 49 **blind, one eye** blind : 19.

Died: at least 4. **Probably died** : at least 5.

127 **girls** under 16 years of age with Xerophthalmia, 32 **blind, one eye** blind : 14.

Table 15. Xerophthalmia cases seen at Undaan, Surabaja, 1955-1964, by sex and month of examination

	Ja.	Feb.	Mrc.	Apr.	May.	Jun.	Jul.	Aug.	Sep.	Oct.	Nov.	Dec.	Total	Adults
1955 children	213	170	164	169	169	183	194	192	172	214	184	170	2194	11122
Xerophthalmia	16	10	22	30	33	43	43	47	42	54	18	11	371	
Percentage	7½%	6%	13%	17%	19%	23%	23%	23%	24%	25%	9%	5%	17%	
1956 children	225	207	191	170	186	213	198	225	198	211	177	204	2405	11410.
Xerophthalmia	14	16	20	25	42	29	33	35	23	25	11	8	282	
Percentage	6%	8%	10%	14%	22%	13%	16%	15%	11%	12%	6%	4%	12%	
1957 children	220	160	202	208	210	182	201	246	277	248	238	270	2662	12999.
Xerophthalmia	15	24	21	28	21	20	40	38	31	13	25	10	286	
Percentage	6½%	15%	8%	16%	10%	11%	20%	15%	11%	5%	10%	3½%	11%	
1958 children	293	226	237	196	241	259	309	309	287	309	261	317	3244	14999.
Xerophthalmia	26	23	20	34	40	39	48	59	49	30	27	24	419	
Percentage	9%	11%	8%	17%	16%	15%	15%	19%	17%	9½%	10%	7%	13%	
1959 children	359	240	326	272	322	325	357	335	324	218	294	219	3731	14832.
Xerophthalmia	17	11	20	22	39	37	34	44	40	27	10	13	313	
Percentage	5%	4½%	6%	8%	12%	12%	9½%	13%	13%	12%	3%	6%	8%	
1960 children	385	290	211	307	307	276	356	376	389	333	301	286	3717	15501.
Xerophthalmia	23	35	27	47	45	65	52	47	50	29	25	25	470	
Percentage	6%	12%	12%	15%	15%	23%	14%	12%	12%	9%	8%	8%	13%	
1961 children	273	240	225	315	255	282	535	483	521	532	532	480	4573	13347.
Xerophthalmia	20	21	37	32	45	23	44	32	24	15	15	13	321	
Percentage	7½%	8%	15%	10%	17%	8%	8%	6½%	4½%	3%	3%	3%	7%	
1962 children	614	268	434	466	438	463	593	462	467	523	354	265	5357	12411.
Xerophthalmia	15	16	27	32	40	34	36	53	29	19	23	11	335	
Percentage	2½%	6%	6%	7%	9%	7%	6%	11%	6%	3½%	8%	4%	6%	
1962 children	309	370	308	347	343	348	415	303	309	324	312	376	3964	13064.
Xerophthalmia	19	18	25	39	60	55	66	28	35	55	19	27	436	
Percentage	6%	5%	8%	11%	17%	17%	15%	9%	11%	17%	6%	9½%	11%	
1964 children	378	331	308	328	356	443	465	396	398	379	389	299	4469:	12956.
Xerophthalmia	52	32	24	29	28	37	48	39	22	17	20	20	369	
Percentage	11%	9½%	7½%	9½%	8%	8%	10%	10%	5½%	4½%	5%	6½%	8%	

Table 16. Number of Xerophthalmia cases by month of examination, by sex, and as a percentage of the total number of children outpatients. 1959.

Month	Boys						total	Girls					
	X1	%	X2	%	X3	%		X1	%	X2	%	X3	%
Jan.	5	1½%	—	—	5	1½%	180	1	½%	1	½%	4	2½%
Feb.	2	1½%	2	1½%	1	1 %	120	1	1 %	—	1 %	4	3 %
Mrc.	8	5 %	—	—	7	4½%	158	2	1 %	—		2	1 %
Apr.	9	6½%	11	1 %	1	1 %	136	7	5 %	—	—	3	3½%
May	15	9 %	2	1 %	7	4½%	161	10	6½%	1	½%	5	3 %
Jun.	13	8 %	1	½%	10	7 %	157	9	5½%	—	—	3	2 %
Jul.	9	5 %	—	—	8	4½%	178	9	5 %	2	1 %	5	2½%
Aug.	17	10 %	—	—	8	4½%	168	15	9 %	3	1½%	—	—
Sep.	18	11 %	2	1 %	9	5½%	162	11	6½%	—	—	4	2½%
Oct.	9	8 %	2	1½%	5	4½%	109	3	2½%	1	1 %	6	5½%
Nov.	—	—	—	—	3	2 %	147	—	—		—	7	4½%
Dec.	1	1 %	—	—	5	4½%	110	1	1 %	—	—	6	5½%
Total	106		10		69			69		9		49	

Table 16 a. Xerophthalmia 1959, by age and degree.

		—1	—2	—3	—4	—5	—6	6 and over
Boys	X1	—	11	43	28	12	4	14
	X2	2	2	5	1	—	—	—
	X3	12	9	26	14	4	1	4
total		14	22	74	43	16	5	18
Girls	X1	1	14	18	24	7	2	3
	X2	—	1	7	1	—	—	—
	X3	6	13	12	7	6	4	1
total		7	28	37	32	13	6	4

1960. Total outpatients : 15501 adults/3717 children : 18% children.
Blind children : 273 **boys** under 16 years of age with Xerophthalmia,
19 **blind** by Xerophthalmia, 21 blind **in one eye**
186 **girls**
11 **blind** 18 blind **in one eye**

Died: at least 2.

Table 17. Xerophthalmia cases seen in 1960, by month of examination, percentages of total children seen during each month.

Month	Boys						Total	Girls					
	X1	%	X2	%	X3	%		X1	%	X2	%	X3	%
Jan.	4	2%	6	3 %	3	1½%	193	4	2 %	2	1 %	4	2 %
Feb.	9	6 %	1	½%	7	5 %	145	13	9 %	2	2½%	3	2 %
Mrc.	11	10%	1	1 %	6	5½%	106	7	6½%	1	1 %	1	1 %
Apr.	14	9 %	1	½%	11	7 %	153	13	8½%	2	1½%	6	4 %
May	19	12%	3	2 %	4	2½%	153	10	6½%	5	3 %	2	1½%
Jun.	19	14%	2	1½%	6	4 %	138	20	15 %	6	4 %	6	4 %
Jul.	19	11%	2	1 %	9	5 %	178	15	8 %	2	1 %	5	3 %
Aug.	22	12%	4	2½%	5	2½%	188	9	4½%	4	2 %	2	1 %
Sep.	23	13%	1	½%	11	5½%	190	9	4½%	2	1 %	4	2 %
Oct.	10	6%	5	3 %	5	3 %	166	4	2½%	1	½%	3	2 %
Nov.	7	5%	3	2 %	7	4½%	150	2	1½%	3	2 %	4	2½%
Dec.	9	6%	2	1½%	4	3 %	143	7	4 %	3	2 %	1	½%
Total	172		32		78			114		33		41	

Table 17 a. Xerophtahalmia 1960, by age and degree.

age in years		—1	—2	—3	—4	—5	—6	and over
Boys,	X0, 1	3	21	62	42	17	9	17
	X2,	6	10	8	5	1	1	—
	X3	15	9	26	14	7	4	1
Total		24	40	96	61	25	14	18
Girls	X0, 1	2	12	52	22	12	9	5
	X2	4	7	13	5	1	2	1
	X3	5	8	16	7	3	—	1
Total		11	27	81	34	16	11	7

1961. Total outpatients : 13347 adults/4673 children : 25 % children.
Blind children : 191 **boys** with Xerophthalmia, 14 **blind** by Xerophthalmia.
one eye blind 9
128 **girls** 12 blind
One eye blind 17

Died : at least 14. probably died : 2.

Table 18. Xerophthalmia cases seen in 1961, by month of examination, percentage of total children seen during the month.

Month	Boys						GIrls						
	X1	%	X2	%	X3	%	Total	X1	%	X2	%	X3	%
Jan.	5	$3\frac{1}{2}$%	3	2 %	1	$\frac{1}{2}$%	136	4	3 %	3	2 %	4	3 %
Feb.	4	3 %	1	1 %	4	3 %	120	4	3 %	2	$1\frac{1}{2}$%	2	$1\frac{1}{2}$%
Mrch.	15	13 %	6	5 %	2	2 %	112	11	10 %	—	—	1	1 %
Apr.	15	$9\frac{1}{2}$%	4	$2\frac{1}{2}$%	4	$2\frac{1}{2}$%	157	9	$6\frac{1}{2}$%	—	—	5	3 %
May	17	13 %	6	$4\frac{1}{2}$%	6	$4\frac{1}{2}$%	127	6	$4\frac{1}{2}$%	4	3 %	6	$4\frac{1}{2}$%
Jun.	10	7 %	1	$\frac{1}{2}$%	2	$1\frac{1}{2}$%	141	5	$3\frac{1}{2}$%	1	$\frac{1}{2}$%	3	2 %
Jul.	15	$5\frac{1}{2}$%	4	$1\frac{1}{2}$%	7	$2\frac{1}{2}$%	267	12	$4\frac{1}{2}$%	—	—	6	2 %
Agus.	15	6 %	1	$\frac{1}{2}$%	3	1 %	241	10	4 %	1	$\frac{1}{2}$%	2	1 %
Sep.	6	$2\frac{1}{2}$%	1	$\frac{1}{2}$%	4	$1\frac{1}{2}$%	260	9	$4\frac{1}{2}$%	1	$\frac{1}{2}$%	2	1 %
Oct.	3	1 %	4	$1\frac{1}{2}$%	1	$\frac{1}{2}$%	266	3	1 %	3	1 %	1	$\frac{1}{2}$%
Nov.	7	$2\frac{1}{2}$%	4	$1\frac{1}{2}$%	3	1 %	266	1	$\frac{1}{2}$%	—	—	1	$\frac{1}{2}$%
Dec.	1	$\frac{1}{2}$%	2	1 %	4	$1\frac{1}{2}$%	240	—	—	1	$\frac{1}{2}$%	5	2 %
Total	113		36		14		74			16		38	

Table 18 a. Xerophthalmia 1961, by age and degree.

	—1	—2	—3	—4	—5	—6	and over
Boys, X0, 1	1	11	41	34	16	3	10
X2	4	11	10	8	2	1	1
X3	8	6	14	9	2	1	1
Total	13	28	64	51	20	5	12
Girls, X0, 1	3	16	24	23	7	1	—
X2	—	4	5	5	1	1	—
X3	7	13	9	6	2	—	1
Total	10	33	38	34	10	2	1

1962 Total outpatients: 12411 adults/5357 children: 30% children.
Blind children : 198 **boys** with Xerophthalmia, 12 **blind** by Xerophthalmia.

one eye blind : 12
141 **girls** 12 **blind**
one eye blind : 28

Died: at least 7. **probably died**: at least 5.

Table 19. Xerophthalmia cases seen in 1962, by month of examination, percentages of total children seen during the month.

Month	Boys						Total	Girls					
	Xo,1	%	X2	%	X3	%		Xo,1	%	X2	%	X3	%
Jan.	2	1/2%	3	1 %	4	1 1/2%	307	1	1/2%	1	1/2%	4	1 %
Feb.	3	2 %	3	2 %	4	3 %	134	4	3 %	—	—	2	1 1/2%
Mrch.	9	4 %	1	1/2%	2	1 %	217	7	3 %	3	1 1/2%	4	2 %
Apr.	16	6 1/2%	1	1/2%	1	1/2%	233	9	4 %	3	1 %	2	1 %
May	18	8 %	1	1/2%	5	2 %	219	11	5 %	4	2 %	3	1 1/2%
Jun.	18	5 1/2%	3	1 %	3	1 %	331	7	2 %	1	1/2%	1	1/2%
Jul.	21	6 %	1	1/2%	1	1/2%	297	9	3 %	2	1/2%	3	1 %
Aug.	24	10 %	4	1 1/2%	1	1/2%	231	19	7 1/2%	5	2 %	2	1 %
Sep.	14	6 %	1	1/2%	2	1 %	233	10	4 1/2%	—	—	1	1/2%
Oct.	7	2 1/2%	2	1/2%	1	1/2%	261	8	3 %	—	—	3	1/2%
Nov.	9	5 %	1	1/2%	5	3 %	172	4	3 %	1	1/2%	3	1 1/2%
Dec.	5	3 1/2%	2	1 1/2%	—	—	132	3	2 %	—	—	1	1/2%
Total	146		23		29			92		20		29	

Table 19 a. Xerophthalmia 1962, by age and degree.

age in yrs		—1	—2	—3	—4	—5	—6	and over
Boys,	X0, 1	4	17	59	34	14	7	13
	X2	4	4	11	2	1	—	1
	X3	6	7	6	6	1	2	1
Total		14	28	76	42	16	9	15
Girls,	X0, 1	3	9	36	27	7	5	6
	X2	2	3	11	2	2	—	—
	X3	5	3	12	7	2	—	—
Total		10	15	59	36	11	5	6

1963. Total outpatients for the whole years 13064 adults/2964 children : 23%. (Because of leave abroad, this years is not completely reported).

Table 20. Xerophthalmia cases seen 1/1'63-1/8'63, by month of examination, percentages of total children seen during the month.

Month	Boys						Total	Girls					
	X0,1	%	X2	%	X3	%		X0,1	%	X2	%	X3	%
Jan.	9	$5^1/_2\%$	—	—	2	1 %	154	3	2 %	3	2 %	1	$^1/_2\%$
Feb.	11	6 %	1	$^1/_2\%$	1	$^1/_2\%$	185	8	4 %	1	$^1/_2\%$	2	1 %
Mrch.	10	$6^1/_2\%$	3	2 %	6	4 %	154	1	$^1/_2\%$	3	2 %	5	3 %
Apr.	21	12 %	3	$1^1/_2\%$	3	$1^1/_2\%$	173	9	4 %	1	$^1/_2\%$	4	$2^1/_2\%$
May	30	17 %	3	$1^1/_2\%$	9	5 %	171	23	13 %	—	—	1	$^1/_2\%$
Jun.	34	19 %	6	$3^1/_2\%$	6	$3^1/_2\%$	174	16	10 %	1	$^1/_2\%$	2	1 %
Jul.	32	16 %	7	3 %	6	3 %	207	20	$9^1/_2\%$	2	1 %	5	$2^1/_2\%$
Total	147		23		33			80		11		19	

Table 20 a. Xerophtahalmia 1/1'63-1/8'63 by age and degree.

age in years	—1	—2	—3	—4	—5	—6	and over
Boys, X0, 1	1	6	52	44	27	4	13
X2	1	1	14	5	2	—	—
X3	4	3	11	6	8	—	1
Total	6	10	77	55	37	4	14
Girls, X0, 1	—	3	36	28	9	2	2
X2	—	4	4	3	—	—	—
X3	1	3	5	7	2	1	—
Total	1	10	45	38	11	3	2
Total							

Comment: From the figures for Xerophthalmia during 1/1'59-1/8'63 there appeared several trends :

1. A higher number of cases of Xerophthalmia in boys than in girls. Although in general the number of boys and girls in the Outpatients' Dept. is more or less the same, there is consistently a higher number of boys than of girls suffering from Xerophthalmia, especially in Xo, 1. Fig. I.

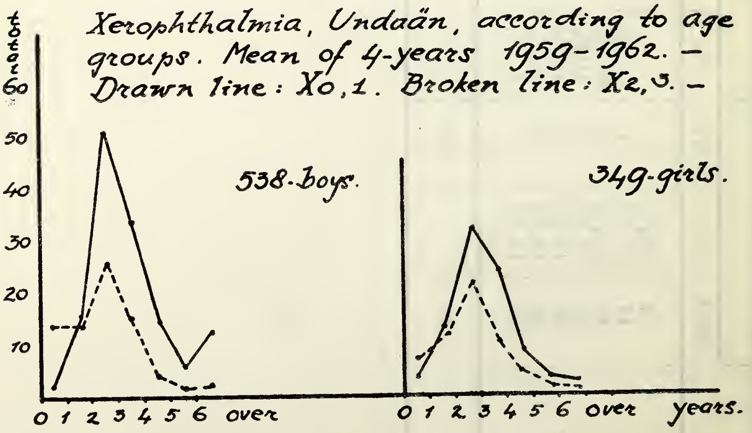


Fig. I.

2. In each year there is a seasonal peak in the frequency of Xerophthalmia, from April through August. This peak is clearest to be seen in the Xo, 1 group, far less in X2 and X3, more in boys than in girls. Fig: K.

Xerophthalmia, Undaän, seasonal peak as a percentage of total children outpatients under 8-yrs. of age per month. 4-yrs 1959-1962. Drawn line: Xo, 1 Broken line: X 2,3.—

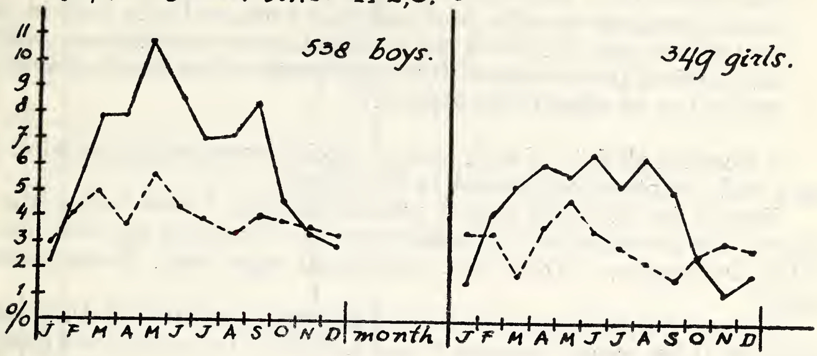


Fig. K.

3. The number of cases who died, is not certain : those who lived in the city of Surabaja were visited at home for a check-up if they were blind, but we know nothing about the fate of the children who came from outside the town. Anyway, mortality was rather high, although we could admit a number of children to the Research Institute for treatment.
4. The number of eyes lost is also rather high, making Xerophthalmia in this region the most important blinding disease in children.

All of these facts warranted a more intensive study, which was executed between 1/8'65 and 1/8'67, observing all Xerophthalmia cases who came to the Outpatients' Dept. during that time, i.e. during 2 consecutive years.

Plan for Xerophthalmia Study.

It was the purpose of the study to ascertain as many facts about Xerophthalmia as it is seen in and around Surabaja as possible with the means at our disposal. The patients came from rather a large area around Surabaja as well as from the city itself, whereas from outside the city often the degree of Xerophthalmia was more serious on an average than that seen in children from the city itself. This is understandable, as help in a small village is often inadequate, and the distance to the city rather far.

The points I planned to investigate were :

1. Incidence in relation to : age, sex, nutritional status, season.
2. Etiology : history of previous illnesses, diet, family composition, social factors (adoption, poverty, inadequate housing etc.)
3. Monocular cases of X2 and X3 : smears were taken from the conjunctiva frome ach eye, to ascertain whether there were differences in bacterial flora.

4. Vitamin A-level of blood serum : in a small number of cases vitamin A-levels could be determined in the laboratory of the Research Institute.
5. Home visits : a social worker trained as such with extra training in this special field, visited many of the patients, and all of those who came during the months June and July 1966, and who lived in the city of Surabaja. She filled out standard reports on the home visits, and advised parents especially of the more serious cases as well on medical as on educational sapects.

I recorded all data of each case on special forms ,which included all signs and symptoms we wished to investigate.

Except for the short time I was on holiday, I saw during the 2 years of the investigation all children under 10 years of age, who came to the Outpagtients' Dept. and selected all cases with Xeropthalmia myself.

Many of the patients were seen by the social pediatrician dr. Gondosoebarijo at her weekly sessions in our hospital. Data on general examination, like liver enlargement, lung ailments etc. were collected by her. The external signs related to malnutrition (condition of skin, hair, eyelashes, etc.) I recorded myself.

III. **Results of the Study on Xerophthalmia** 1/8'65—1/8'67.

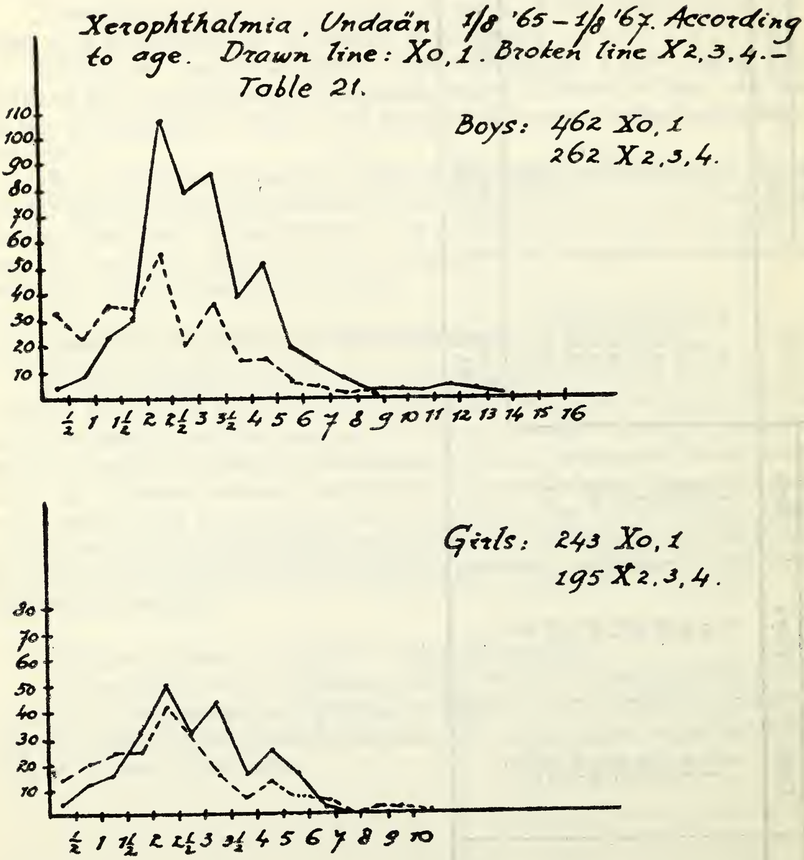
It was possible to examine 1149 children suffering from Xerophthalmia during the 2 year-period, all of them consecutive cases. From our data we ascertained the location of certain foci where Xerophthalmia was more prevalent in the city of Surabaja. In one of these foci there was carried out a house-to-house survey for Xerophthalmia (Hoepoediono) of which the results have not yet been published. Our home visitor reported on the frequency of Xerophthalmia in certain quarters of the town, mostly parts of the town where housing was provisory only and mud and rubbish were plentiful.

A. Relation of **age** and **degree** of Xerophthalmia.

As had been seen in previous years, severe Xerophthalmia tends to be seen in the younger age groups. In **Table** 21 the relation is given for 2 degrees of severity of Xerophthalmia (X0 and X1 taken together, X2, X3 and X4 taken together).

Age	X0,1		X2,24		Age	X0,1		X2,34	
	Boys	Girls	Boys	Girls		Boys	Girls	Boys	Girls
—½ yr	3	4	32	13	—8 yr	7	—	—	—
—1 yr	8	11	21	20	— 9 yr	1	—	1	1
—1½ yr	23	16	35	23	—10 yr	1	—	—	1
—2 yr	29	31	32	23	—11 yr	1	—	—	—
—2½ yr	106	50	54	41	—12 yr	2	—	—	—
—3 yr	79	30	19	30	—13 yr	1	—	—	—
—3½ yr	84	43	35	17	—14 yr	—	—	—	—
—4 yr	37	15	12	6	—15 yr	—	—	—	—
—5 yr	50	25	13	11	—16 yr	(2)	—	—	—
—6 yr	18	16	5	6	over	(2)	—	—	—
—7 yr	12	2	3	3					
					Total X0,1	462 : 705	243	262 X2,3,4:457	195

For **boys** with **Xo, 1:** 50% of cases is younger than 3 years.
Of **girls** with **X0,1** 55% is younger than 3 years.
Of **boys** with **X2,3,4** 66 is younger than $2^1/_2$ years.
girls **X2,3,4** 61%



Xerophthalmia, Undaän 1/8 '65 – 1/8 '67. According to age. Drawn line: Xo, 1. Broken line X2,3,4. – Table 21.

Boys: 462 Xo, 1
262 X2,3,4.

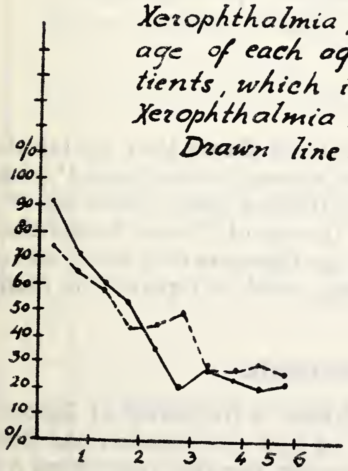
Girls: 243 Xo, 1
195 X2,3,4.

Fig. L.

Figure L presents the figures from table 21, and shows a distinct peak in frequency of Xerophthalmia Xo, 1 as well in boys as in girls. For X2, 3, 4, there is a higher frequency at a lower age, rising to a less high peak than Xo, 1, but declining earlier, after 3½ year age-group.

Table 22. Percentage of each age group which is suffering from severe Xerophthalmia.

Age	—½	—1	—1½	—2	—2½	—3	—3½	—4	—5	—6	year
Boys	19%	72%	60%	52%	34%	19%	19%	24%	20%	21%	is X2, 3, 4.
Girls	76%	64%	59%	42%	45%	50%	28%	28%	30%	27%	is X3, 3, 4.



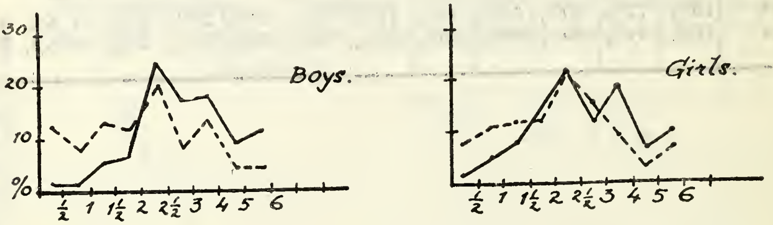
Xerophthalmia, Undaän 1/8 '65 – 1/8 '67. Percentage of each age group of xerophthalmiapatients, which is suffering from serious (X2,3,4) Xerophthalmia. Table ......
Drawn line: boys. Broken line: girls.

Fig. M.

Figure M is representing the data from table 22. It is clear that from infants and very young children with Xerophthalmia a very high percentage has severe Xerophthalmia, often blinding, often also fatal. This group contains many children who were not breastfed, which is dangerous, as van Stockum already pointed out (482).

Table 23. Age groups as a percentage of total of each degree of Xerophthalmia for each sex. (cases over 6 yrs. old included in totals).

age	—$^1/_2$	—1	—1$^1/_2$	—2	—2$^1/_2$	—3	—4	—5	—6	years
Boys, X0,1	0.6%	1.7%	5%	6%	24%	17%	18%	8%	11%	
X2,3,4	12 %	8%	13%	12%	20%	7%	13%	4%	4%	
Girls X0,1	1.6%	4.5%	6.5%	12%	20%	12%	18%	6%	10%	
X2,3,4	6.6%	10%	11%	11%	21%	15%	9%	3%	6%	



Xerophthalmia, Undaän, 1/8-'65 – 1/8-'67. Degrees of Xerophthalmia as a percentage of each age group. Boys and girls seperate. Xo,1: — X2,3,4: ----
(Table - 23.)

Fig. N.

Figure. N showing the data from table 23 shows that by far the largest percentage of Xerophthalmia cases is seen between ages 1$^1/_2$ and 3$^1/_2$ years, as well in boys as in girls, with a distinct peak around age 2$^1/_2$ years, in Xo, 1 as well as in X2, 3, 4. Over the age of 6 years Xerophthalmia becomes much rarer, over 10 years of age there are only rare cases of Xo, 1 in boys, not in girls. This is a finding which is reported by many investigators.

Seasonal Peak of Xerophthalmia at Surabaja.

In the recording of the seasonal variation in frequency of Xerophthalmia in former years, only the month of first examination had been taken into account. In the investigation during the two years from August 1965 till August 1967, I recorded not only the month of first examination, but also the month of beginning of the eye complaints. Also cases which had healed with corneal scarring I included if they had been active not longer ago than 3 months (X4). If a case was still active, it was also included if it had begun longer than 3 months ago. If a case which had been registered before in the period under study, but which had a recurrence during this period, was seen, it was placed in column R (for Recurrence). In the following Tables 24 and 24a the column marked I gives the number of cases which came for first examination, the column marked II gives the corrected month of beginning of the eye complaint. The months June and July '65 show only the Xerophthalmia cases seen for the first time during the period of study, not all of the Xerophthalmia cases (which would be a far larger number).

Table 24. Xerophthalmia by month of examination (I) and by month of beginning (II). R : recurrences in cases seen before. Boys.

			I					II			
Month		R	X0	X1	X2	X2	X4	X0,1	R	X2,3,4	Total
1965.											
Jun.	—	—	—	3	—	1	—	3	—	1	4
Jul.	—	—	—	5	—	—	—	5	—	—	5
Aug.	37	—	4	21	1	8	—	25	—	9	34
Sep.	25	—	1	12	3	7	—	13	—	10	23
Oct.	19	—	1	11	3	11	—	12	—	14	26
Nov.	16	—	1	7	—	5	—	8	—	5	13
Dec.	16	—	—	6	3	5	—	6	—	8	14
1966.											
Jan.	11	3	1	5	1	5	—	6	3	6	12 (15)
Feb.	29	—	1	20	—	7	2	21	—	9	30
Mrc.	27	—	—	14	4	8	1	14	—	13	27
Apr.	36	2	4	16	4	14	—	20	2	18	38 (40)
May	43	—	4	32	2	10	—	36	—	12	48
Jun.	58	2	2	35	6	12	—	37	2	18	55 (57)
Jul.	46	3	4	31	3	12	—	35	3	15	50 (53)
Tot.	363	10	23	218	30	105	3	241	10	138	379 (389) 1 year 1/8'65-1/8'66.
Aug.	37	2	5	20	2	2	1	25	2	5	30 (32)
Sep.	31	1	2	20	2	8	1	22	1	11	33 (34)
Oct.	31	2	1	21	1	4	—	22	2	5	27 (29)
Nov.	10	1	1	2	—	5	—	3	1	5	8 (9)
Dec.	15	—	—	10	3	5	—	10	—	8	18
1967.											
Jan.	21	—	—	6	5	5	—	6	—	10	16
Feb.	14	1	1	8	1	3	—	9	1	4	13 (14)
Mrc.	25	1	—	13	5	10	—	13	1	15	28 (29)
Apr.	27	3	2	18	3	8	—	20	3	11	31 (34)
May	53	3	7	35	5	11	—	42	3	16	58 (61)
Jun.	50	2	3	23	5	16	1	26	2	22	48 (50)
Jul.	47	3	3	19	5	6	—	22	3	13	35 (38)
Tot.	361	19	25	195	37	85	3	220	19	125	345 (364) 1 year, 1/8'66-1/8'67.
Tot.	724	29	48	413	67	190	6	461	29	263	724 (753) 2 years, 1/8'65-1/8'67.

Table 24 a. GIRLS.

I								II			
Month	R		X0	X1	X2	X3	X4	X0,7	R	X2,3,4	Total † R)
1965.											
Jun.	—	—	—	—	—	1	—	—	—	1	1
Jul.	—	—	—	3	—	2	—	3	—	2	5
Aug.	20	—	—	10	—	6	—	10	—	6	16
Sep.	11	—	—	4	3	5	—	4	—	8	12
Oct.	14	—	—	11	2	3	—	11	—	5	16
Nov.	8	—	—	4	1	3	—	4	—	4	8
Dec.	12	—	—	2	3	9	—	2	—	12	14
1966.											
Jan.	13	—	—	3	2	9	—	3	—	11	14
Feb.	17	2	1	8	2	7	—	9	2	9	18 (20)
Mrh.	17	1	1	10	2	11	—	11	1	13	24 (25)
Apr.	27	—	3	6	3	9	—	9	—	12	21
May	30	—	2	16	2	7	—	18	—	9	27
Jun.	28	—	1	19	2	8	1	20	—	11	31
Jul.	28	—	2	13	5	5	—	15	—	10	25
Tot.	235	3	10	109	27	85	1	119	3	113	232 (235) 1 year, 1/8'65-1/8'66
Aug.	23	1	3	12	2	7	—	15	1	9	24 (25)
Sep.	22	2	1	13	—	5	—	14	2	5	19 (21)
Oct.	17	—	2	8	2	5	1	10	—	8	18
Nov.	8	—	—	4	1	3	—	4	—	4	8
Dec.	13	—	—	5	1	6	—	5	—	7	12
1967.											
Jan.	6	—	—	2	—	5	—	2	—	5	7
Feb.	16	1	1	11	—	8	—	12	1	8	20 (21)
Mrh.	13	—	—	5	2	6	—	5	—	8	13
Apr.	24	—	4	13	2	6	—	17	—	8	25
May	31	—	2	16	—	8	1	18	—	9	27
Jun.	20	—	1	13	1	5	—	14	—	6	20
Jul.	20	2	2	7	1	3	2	9	2	6	15 (17)
Tot.	213	6	16	109	12	67	4	125	6	83	208 (214) 1 year 1/8 66-1/8'67.
Tot.	438	9	26	218	39	152	5	244	9	196	440 (449) 2 years, 1/8'65-1/8'67.

The total number in the "corrected" column II is higher than that in column I, as also a number of cases which were seen for the first time AFTER the period of study were included.

The trends which can be discovered or confirmed from Table 24 and 24a are as follows :

1. More boys than girls suffer from Xerophthalmia in this region.
2. This difference is more distinct in the Xo, 1 degree of Xerophthalmia.
3. Xerophthalmia tends to be severe if it strikes before the age of 2 years.
4. In Xo, 1 cases there is a distinct peak in frequency starting in April, culminating in June-July, declining again to a low from November till March. This peak is higher in boys than in girls.
5. For severe Xerophthalmia (X2, 3, 4) there is a rise in frequency in the same months as in light Xerophthalmia (Xo, 1), but not nearly as high, and more distinct in boys.
6. Recurrences (R) were generally more frequent in the "Peak" season.
7. Nightblindness only (Xo) was more frequent in the "Peak" season, for boys and girls.
8. Recurrences in boys are relatively as frequent as in girls.
9. Comparison between period 1959-1960 and 1965-67 (2 periods of 2 years) : there is a trend for severe Xerophthalmia (X2, 3, 4) to INCREASE in percentage at an early age :

   In the earlier two year-period 35% of **boys** with **X2, 3** were **under 2 yrs**. of age.

   In the later two year-period 46%

   In the earlier two year-period 32% of **girls** with **X2, 3** were **under 2 yrs**. of age.

   In the later two year-period 40%.

Number of **Xerophthalmia** cases compared to **number of children outpatients.** All children under 8 years of age who came to the Outpatients Dept. between Aug. 1st 1965 and Jan. 1st 1967 were counted and the total per month was compared with the total number of Xerophthalmia cases of all degrees in the same month. The correction for the real beginning of Xerophthalmia was not applied in this comparison.

Table. 25. Xerophthalmia cases as a percentage of all children outpatients under 8 years of age, by month and by sex.

	Boys						Girls					
Month	total	Xer.	Recurr.	X : %	non-xer	%(+R)	total	Xer.	Recurr.	X :%	Non-xer	%(+R)
1965.												
Aug.	106	37	—	35%	69		89	20	—	22%	69	
Sep.	103	25	—	24%	78		82	11	—	13%	71	
Oct.	92	19	—	20%	73		83	14	—	17%	69	
Nov.	67	16	—	24%	51		70	8	—	11%	62	
Dec.	73	16	—	22%	57		59	12	—	20%	47	
1966.												
Jan.	67	11	3	16%	56	(20%)	81	13	—	16%	68	
Feb.	81	29	—	35%	52		77	17	2	22%	60	(23%)
Mrh.	78	27	—	34%	51		60	17	1	28%	43	(19%)
Apr.	85	36	2	42%	49	(43%)	75	27	—	36%	48	
May	104	43	—	41%	61		78	30	—	38%	48	
Jun.	102	58	2	56%	44	(57%)	76	28	—	37%	48	
Jul.	106	46	3	43%	60	(45%)	83	28	—	34%	55	
Aug.	111	37	2	33%	74	(34%)	99	23	1	23%	76	(24%)
Sep.	95	31	1	32%	64	(33%)	98	22	2	22%	76	(23%)
Oct.	97	31	2	32%	66	(33%)	86	17	—	19%	69	
Nov.	74	10	1	14%	64	(14%)	66	8	—	12%	58	
Dec.	60	15	—	25%	45		55	13	—	23%	42	
Total 1/8'65-1/1'67	1501	487	16		1014		1317	308	6		1009	
total 1966 only.	1060	374			686		934	243			691	

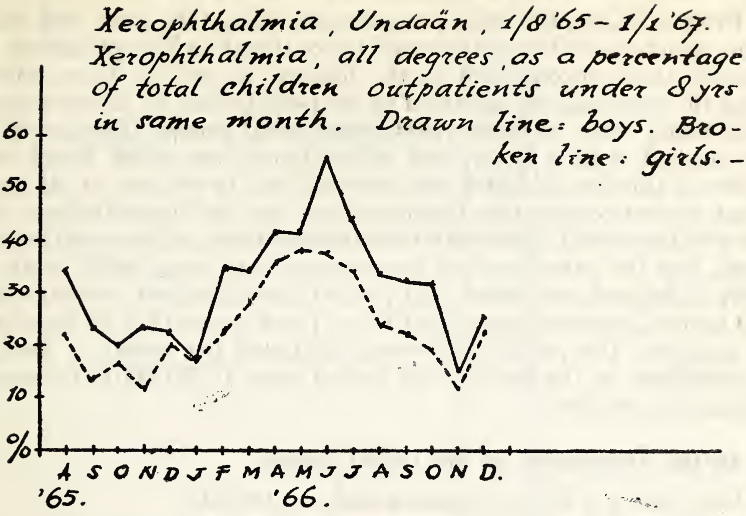


Fig. O.

In figure O the data of table 25 are represented by a curve.

The following conclusions can be drawn:

1. Of our boy outpatients under the age of 8 year 32% suffer from Xerophthalmia, or 35% if the percentage is calculated only for the year 1966. For girls the percentage is 23% over the whole period, 26% for the year 1966 only. The whole period concerns two "off seasons" and one "season", so it is more correct to calculate the overall percentage for a one year-period only, i.e. for 1966. In season Xerophthalmia can even reach a monthly percentage of 56% in boys of all boy patients, if recurrences are taken into account even 57%. For girls the top percentage in one month reaches 38%.

2. The difference between the total number of boy patients (1501) and girl patients (1317) is the same as the difference between the total number of boys with Xerophthalmia (487) and girls with Xerophthalmia (308). This means that the fact that more boys were seen is entirely due to the fact that more boys suffer from Xerophthalmia, for all other eye diseases there was no difference in number between boys and girls!

3. From 2. can be concluded that boys are not better attended by their parents than girls, as interest in all other eye diseases is evenly divided between boys and girls, i.e. it can be supposed that the parents will bring their children with eye complaint, regardless whether boys or girls. A parental preference for boys cannot be construed from these figures.

Causal factors in Xerophthalmia, Undaan, 1966.

From all Xerophthalmia patients seen during the year 1966 various factors which could have influenced the outbreak of Xerophthalmia were recorded, those encountered in the history, as well as those collected during the examination (also that of the pediatrician dr. Gondosoebarijo) and in many cases on home visits of the social worker. The factors were each marked with a letter, and calculations were made based on the number of times each factor was encountered. It was not at all unusual to find several cooperating factors in one case of Xerophthalmia, e.g. a child which received a diet with too little carotene, and also had intestinal worms. For the calculation of percentages only cases which were completely examined were used, and patients were divided as before in the usual groups, boys and girls, light (Xo, 1) and severe (X2, 3) Xerophthalmia separate. The percentages were calculated per month. I used only the year 1966, as the data on the period from 1/8'65 till/11'66 were not sufficiently complete.

Key to the designation of the causal factors.

A. Diet with too little vitamin A and carotenoids.
B. Infestation with Ascaris prior to outbreak of Xerophthalmia.
D. Diarrhoeal disease, prior to outbreak of Xerophthalmia.
E. Tuberculosis (either pulmonary or glandular, usually Mantoux test positive).
F. Other infectious disease (Measles, chickenpox, parotitis, otitis media, bronchitis, pyodermia etc.).
G. Twins.
I. Icterus.
K. Artificial feeding.
L. Younger baby present in family.
M. Poverty.
P. Prematurity.
R. Family moves back and forth between city and village (semi-urbanized).
S. Social factor (orphan or semi-orphan, parents divorced, mother ill, working outside the home, dead, very stupid etc.).
T. Adopted child (often by childless relative).
V. House and/or immediate surroundings very dirty (often only the surroundings by inadequate garbage disposal, flooding).
?.? No etiological factor encountered : seemingly healthy child.
— Insufficient data.

Comment : as factors which would possibly be influenced by the season, especially factors A, D and F are to be taken into account (resp. diet deficient in A-sources. Diarrhoeal disease and infectious diseases). To make sure whether other factors could be influenced by the season they were calculated also, but only in factors A/F was there a certain relation discernible, whereas all other factors showed a very irregular pattern, during the year, as was to be expected. I calculated different combinations of several groups of patients taken together, also for 1966 only.

Table 26. number of times the causal factors were found in BOYS, Xo, 1, by month.

Factor	Jan.	Feb.	Mrh.	Apr.	May	Jun.	Jul.	Aug.	Sep.	Oct.	Nov.	Dec.	Total
A	—	4	4	6	13	26	21	16	14	16	1	5	126
B	2	7	5	9	3	18	15	16	6	5	2	3	93
D	5	10	9	6	11	19	14	17	11	9	2	6	129
E	—	5	1	—	3	14	13	18	4	8	—	1	67
F	3	9	1	9	12	8	7	7	7	13	1	2	79
I	—	1	—	—	—	—	1	—	—	—	—	—	2
K	—	—	1	—	—	3	—	1	—	2	—	—	7
L	—	4	2	4	10	9	15	11	8	5	1	4	73
M	—	1	—	—	—	3	4	3	—	1	—	1	13
P	—	—	—	—	—	1	—	—	—	—	—	—	1
R	—	1	—	1	—	1	3	1	—	—	—	—	7
S	—	3	1	—	—	11	4	1	1	2	—	—	23
T	—	2	—	—	—	2	—	1	—	—	—	—	5
V	—	2	—	—	—	10	14	4	1	—	—	1	32
??	—	3	2	—	—	2	—	—	—	1	—	—	8
—	—	—	—	—	3	—	1	—	1	1	—	—	6
	6	22	14	18	34	38	31	30	21	26	4	8	252 total number of patients.

Table 26a. Percentages of factors A/F for BOYS, Xo, 1,

A in %	—	18	28	33	42	68	70	53	70	64	25	62
B in %	33	32	35	50	16	47	50	53	30	20	40	37
D in %	83	45	63	33	35	50	46	56	55	36	50	75
E in %	—	22	7	—	9	36	43	60	20	32	—	12
F in %	50	40	7	50	38	21	23	23	35	52	25	25

Table 27. Causal factors in BOYS, X2, 3, 4, 1966, by month.

Factor	Jan.	Feb.	Mrh.	Apr.	May.	Jun.	Jul.	Aug.	Sep.	Oct.	Nov.	Dec.	Total
A	1	5	2	5	4	10	6	2	7	—	3	4	49
B	2	4	2	5	1	5	2	2	—	—	—	3	25
D	2	2	5	7	3	7	7	1	5	1	—	4	44
E	—	2	2	1	2	6	6	1	1	—	1	2	24
F	2	7	6	11	5	13	7	3	3	2	2	2	63
G	—	—	—	—	—	—	1	—	—	—	1	—	2
K	—	2	5	6	—	4	—	1	6	—	1	3	28
L	—	3	3	2	1	3	3	2	3	—	1	1	22
M	—	—	1	1	1	7	2	—	2	—	—	—	14
P	—	—	1	—	—	1	—	—	—	—	—	—	2
R	—	1	1	2	—	4	—	—	1	—	—	—	9
S	—	—	2	3	2	9	1	—	4	—	1	1	23
T	—	—	1	1	—	—	—	—	—	—	—	1	3
V	—	—	—	—	—	5	4	1	3	—	—	—	13
——	—	—	—	—	1	—	3	1	1	—	—	—	6
Total	5	8	13	18	10	21	15	7	10	3	6	7	123 number of patients.

Table 27a. Percentages of causal factors A/F in BOYS X2, 3, 4. by month.

A in %	20	62	15	27	44	47	50	33	77	—	50	57
B in %	40	50	15	28	11	23	16	33	—	—	—	28
D in %	40	25	38	38	33	33	58	16	55	33	—	57
E in %	—	25	15	5%	22	28	50	16	11	—	16	28
F in %	40	87	45	63	55	61	58	50	33	66	33	28

Table 28. Causal factors in GIRLS, Xo, 1. 1966.

Factor	Jan.	Feb.	Mh.	Apr.	May	Jun.	Jul.	Aug.	Sep.	Oct.	Nov.	Dec.	Total
A	—	3	3	5	3	14	13	6	5	6	3	4	65
B	1	3	3	3	5	8	15	7	5	3	1	1	55
D	2	3	2	1	9	9	8	8	11	3	2	1	59
E	—	1	—	1	3	6	9	2	7	2	—	—	31
F	3	3	1	4	8	2	3	5	1	4	1	2	37
G	—	—	—	—	—	—	1	—	—	—	—	—	1
I	—	—	—	1	—	—	—	—	—	—	—	—	1
K	1	—	2	1	1	1	—	1	—	1	1	—	9
L	—	1	1	1	4	3	5	3	5	3	—	—	26
M	—	1	2	—	—	4	5	—	1	—	—	—	13
R	—	—	1	—	1	1	—	1	—	—	—	—	4
S	—	1	3	—	2	4	3	2	4	2	—	1	22
T	—	—	—	—	2	—	—	—	—	1	—	1	4
V	—	2	3	—	—	7	6	—	—	—	—	—	18
??	—	—	1	1	1	—	1	—	1	—	—	—	5
——	—	—	—	—	—	—	—	—	1	—	—	—	1
	4	8	7	11	19	17	20	12	18	10	3	5	134 number of patients.

Table 28a. Percentages of causal factors A/F in GIRLS, Xo, 1. 1966.

A in %	—	37	43	45	15	82	65	50	30	60	100	80
B in %	25	37	43	27	26	47	75	58	30	30	32	20
D in %	50	37	28	9	47	53	40	75	64	30	66	20
E in %	—	12	—	9	15	35	45	16	41	20	—	—
F in %	75	37	14	36	42	12	15	41	6	40	33	40

Table 29. Number of times the causal factors were seen in **GIRLS, X2**, 3, 4. 1966.

Factor	Jan.	Feb.	Mrh.	Apr.	May	Jun.	Jul.	Aug.	Sep.	Oct.	Nov.	Dec.	Total
A	2	5	3	2	2	6	8	6	1	5	3	6	49
B	5	3	4	7	3	1	3	1	—	1	—	1	30
D	6	2	3	8	3	4	4	5	3	3	—	3	45
E	—	1	1	1	—	3	3	4	—	2	—	1	16
F	7	6	7	9	3	3	2	4	2	5	4	1	57
G	—	—	—	—	—	1	—	—	—	—	—	—	1
K	2	2	2	3	1	3	2	1	—	—	3	3	22
L	2	3	2	2	—	5	1	3	—	2	—	1	21
M	—	—	—	—	1	2	2	—	—	1	1	—	7
P	—	—	—	—	—	1	—	—	—	—	—	—	1
R	—	1	1	4	1	1	—	1	—	—	—	—	9
S	—	—	1	4	1	5	1	3	1	4	3	1	24
T	—	—	1	3	1	2	—	1	1	—	3	1	13
V	1	—	—	1	1	1	5	—	—	1	—	—	10
??	—	—	—	—	—	—	—	—	—	—	—	1	1
——	—	—	—	—	—	—	—	—	1	—	—	—	1
	9	7	9	16	9	11	9	11	4	7	5	8	105 number of patients.

Table 29a. Percentages of causal factors A/F in **GIRLS, X2**, 3, 4.

A in %	22	71	33	12	22	54	88	60	33	71	60	75
B in %	55	43	41	43	33	9	33	10	—	14	—	12
D in %	66	28	33	50	33	45	44	50	100	43	—	37
E in %	—	14	11	6	—	27	33	40	—	28	—	12
F in %	77	85	77	56	77	27	22	40	66	71	80	12

Table 30. Percentages of light Xerophthalmia (Xo, 1) cases, boys and girls together.

Factor	Jan.	Feb.	Mrh.	Apr.	May.	Jun.	Jul.	Aug.	Sep.	Oct.	Nov.	Dec.	
A in %	—	23	33	38	32	72	70	52	51	61	57	43	
B in %	30	33	37	41	20	47	60	54	29	22	42	19	
D in %	70	43	57	24	40	51	44	60	58	34	57	33	
E in %	—	20	4	3	12	37	44	47	29	27	—	4	
F in %	60	40	9	44	40	18	20	28	21	47	28	19	total 386 patients.

Table 30a. Percentages of severe Xerophthalmia (X2, 3, 4,) boys and girls together.

A in %	21	66	22	20	33	50	60	50	66	50	54	71	
B in %	50	46	27	36	22	22	21	18	—	10	—	21	
D in %	57	26	36	45	33	37	57	37	66	40	—	46	
E in %	—	20	13	6	11	28	39	31	8	20	1	21	
F in %	63	86	59	60	66	30	39	43	41	70	54	21	total 228 patients.

Table 30b. Percentages of light and severe Xerophthalmia together, boys an girls.

A in %	21	66	22	20	33	50	60	50	66	50	54	71	
B in %	50	46	27	36	22	22	21	18	—	10	—	21	
D in %	57	26	36	45	37	37	57	37	66	40	—	56	
E in %	—	20	13	6	11	28	39	31	8	20	1	21	
F in %	63	86	59	60	66	30	39	43	31	70	54	21	total 614 patients.

Conclusions : From the Tables 26 through 30 can be seen, that the higher number of light Xerophthalmia cases seen during the middle months of each year, is accompanied by a higher percentage of children who get too little vitamin A-sources with their food (factor A). In the first and last months of the year the Light Xerophthalmia cases are accompanied by a higher frequency of cases with previous Diarrhoeal disease (D). In the same months there is also a higher percentage of children with previous infectious diseases (factor F). In the percentage of children were Xerophthalmia was preceded by Ascaris (factor B) and Tuberculosis (factor E) there is no clear influence of the season.

In serious Xerophthalmia (X2, 3, 4) there is no single factor which shows a higher percentage of occurrence in any months or season, exactly like severe Xerophthalmia itself, which also shows only a slight rise during the months of the peak-season.

The fact which emerges, and which needs an explanation, is that so many children who are in relatively good health otherwise, seem to live on a marginal diet as far as concerns vitamin A-sources, which precisely in the peak season causes manifest Xerophthalmia in a light degree. A possible reason could be that in East Java during the peak season rice is rather plentiful, which possibly combined with a relative scarceness of vegetables in the dry season, induces the parents to give their children more rice and less vegetables. Another causes cooperating with the former one could be the rather strong and uninhibited sunlight in the middle months of the year (See chapter 24). This would possibly explain the preference of Xerophthalmia for boys, who stay outside the house for longer periods than girls. A third factor could be, as has formerly been seen (Patjet-report, see chapter 24) that the division of the food inside the family is supervised badly or not at all, resulting in the fact that small children and especially boys who oftener are absent from home for hours at a time, do get too little of the foodstuffs containing Protein, Fat and Carotenoids. Probably there is a combination of these various factors, which in cooperation with one another cause Xerophthalmia. The factor light has not yet been investigated sufficiently.

Other causal factors operative in triggering the outbreak of Xerophthalmia.

Analysis of factors G/V did not make visible any influence of the season, as could be expected. The relative importance of these factors can be seen from Table 27, giving the percentages for the year 1966 as a whole :

Table 27. Factors G/V as a percentage of the whole number of Xerophthalmia cases, by sex, and by degree.

Factor	Boys	Girls	together	Boys	Girls	Together
		X0,1			X2,3,4	
G	—	—	—	$1^1/_2$%	—	—
K	2.8%	$6^1/_2$%	4%	27%	21%	$22^1/_2$%
L	28%	19%	23%	19%	17%	19%
M	5%	$9^1/_2$%	$6^1/_2$%	$13^1/_2$%	6%	$9^1/_2$%
P	—	—	—	$1^1/_2$%	—	—
R	2.8%	3%	3%	7%	$8^1/_2$%	8%
S	9%	$16^1/_2$%	$11^1/_2$%	19%	23%	21%
T	2%	3%	2%	$2^1/_2$%	12%	7%
V	13%	$13^1/_2$%	13%	11%	9%	10%
??	3%	$3^1/_2$%	3%	5%	—	1%

Conclusion : From table 27 it is clear that in severe Xerophthalmia (X2, 3, 4) the factor K (artificial feeding) is much more frequent than in light Xerophthalmia, i.e. the well known fact that infants who for one reason or another are not breastfed run a serious risk to develop Xerophthalmia, with ensuing blindness or even death. Often this is not realized neither by the parents who give a child away nor by the people who adopt a child, for which they cannot provide appropriate food. Especially in girls the consequences are still worse than in boys. Related social factors : mother has died, or is working outside the home, or is ill, are also more frequent in severe Xerophthalmia than in the less serious cases. Factor R (going back and forth between town and village) also has a bad influence on the health of the children, as it is not rare, that a small child is staying in town with the father, whereas the mother is somewhere in a village, or a very small child is taken care of by a grandmother who again cannot provide the appropriate food.

The number of times that no causal factor (??) could be elicited except the slight degree of Xerophthalmia or even the serious degree, points to the fact that in some cases, not in very many, only vitamin A-deficiency does occur : this makes it probable that indeed "pure" serious Xerophthalmia does exist in this region. Whether these children have other subclinical signs of malnutrition would call for a more extensive examination than we could provide, e.g. for anemia, serum protein levels etc. These patients run the risk that a slight cold or other infectious disease would trigger severe Xerophthalmia, and that the infectious disease itself also would have a more severe course, precisely because of the vitamin A-deficiency.

The number of times there were insufficient notes (—) has been subtracted from the total of cases for the calculations.

Home visits. A social worker with experience in work with blind children (who already had done the home visiting for the data collected in chapter 26) visited a large number of Xerophthalmia cases at home, and completed standardized reports on her findings. She covered the months June and July 1966 as completely as possible, visiting almost all cases as far as they lived in the city of Surabaja. A small number of cases could not be found, although the time after their first examination at the outpatients' dept. was still only very short. For what reason parents are apt to give a false address is a mystery, but it is not an unusual experience also in other hospitals.

From the total number of Xerophthalmia cases examined at Undaan during June/July 1966, home visits were not made in the following number :

Living outside Surabaja	8 boys (5 Xo, 1 ; 3 : X2,3,4).
	4 girls (2 Xo, 1 ; 2 X2,3,4).
Faulty address	5 boys (3 Xo, 1 ; 2 X2,3,4).
	2 girls (1 Xo, 1 ; 1 X2,3,4).
other reasons	5 boys (2 Xo, 1 ; 2 X2,3,4).
Total not visited	18 boys (8 Xo, 1 ; 10 X2,3,4).
	6 girls (3 Xo, 1 ; 3 X2,3,4).

For June/July 1966 percentages for G/V factors could be calculated using the findings of the home visits in all cases :

Table 28. Percentages of causal factors G/V for Xerophthalmia cases June/ July 1966.

Factor	Boys	Girls	together	boys	girls	together
	Light Xerophthalmia (Xo,1)			Severe Xerophthalmia (X2,3,4)		
G	—	—	—	—	—	—
I	—	—	—	—	—	—
K	4	2	4	4	25	16
L	35	21	30	17	25	20
M	9	24	14	25	20	23
P	$1^1/_2$	—	1	$2^1/_2$	5	$3^1/_2$
R	6	2	$4^1/_2$	8	5	7
S	22	19	20	25	30	29
T	3	—	2	—	10	$3^1/_2$
V	35	35	35	25	30	27
??	2	2	3	—	—	—

Table 28 can be taken as giving the most accurate description of the influence and frequency of the various socio-economic factors involved in the causation of Xerophthalmia, as it covers almost all cases seen during 2 consecutive months.

Factor K: To replace milk, which is scarce and expensive, often all kinds of other foods or pseudo-foods are given, among the latter tea and ricewater, without anything else. It is obvious that a baby cannot grow or stay healthy on tea alone. There are more adequate replacements in use, e.g. SGM, which in the proper dilution is all right, but to "economize" people tend to give a weaker dilution, and of the children who were not breastfed and suffered from Xerophthalmia, a certain number had SGM. It should be realized by whoever prescribes the use of the preparation, that it should be stressed that the correct dilution must be used.

Factor L : Exactly like in Kwashiorkor (which means disease of the deposed boy) Xerophthalmia was also seen in a number of cases where a baby of up to 1 yr. of eage was present in the family. As Wolff pointed out, there is a distinct lack of supervision in the feeding of the child after weaning, resulting in its getting probably sufficient rice, but no other necessary foodstuffs. The child is left to its own devices, and nobody realizes that it needs good food because growing as such is "hard work".

M : Poverty was a rather important factor in severe Xerophthalmia, not so much in light Xerophthalmia.

R : A certain number of Xerophthalmia cases were found in the semi-urbanized part of the population, who often divide their family over town and village, not taking into account the needs of the small children, and not asking for expert advice on their care either. Only after the child by lack of appropriate food has fallen ill it will be brought to a doctor, often after a long delay, caused by the great distance and expense. These cases are often in a deplorable condition.

V : In most cases the house (or hut) itself was reasonably clean, but the surroundings were either swamped or dirty because of garbage piles etc. The housing itself is often "unofficial", and from time to time these dwellings will be razed to clean up the "Wild occupation". See farther chapter 26.

One of the facts our home visitor could ascertain is that there are certain "Xerophthalmia regions" in the town, where nightblindness is so common among young children, that it is not considered alarming at all, "sudah biasa", i.e. "usual" : these children are often not brought to a doctor or dispensary, and heal by themselves, or if they are unlucky they contract an infectious disease, or diarrhoea or Ascaris in the unhygienic surroundings.

An infectious disease will probably have a more serious course, and the lack of good water is another contributing factor. A survey on Xerophthalmia in one of the areas we discovered will be reported elsewhere (Hoepoediono).

DIFFERENT DEGREE OF XEROPHTHAMIA IN BOTH EYES.

Although the corneal lesions in Xerophthalmia are often strikingly symmetrical in both eyes, it can also happen that one eye only shows light signs whereas the other is suffering from severe Xerophthalmia. As Xerophthalmia is clearly caused by a constitutional deficiency, one would expect that the lesions in both eyes were more or less of the same degree. The cause of the difference is most probably external, e.g. a slight trauma, or different bacterial flora, which by secondary infection could be more dangerous on one side.

Slight trauma : As a rule children are carried in the left arm, or if in the slendang also on the left side. To see if this difference in exposure of the right and the left eye was of importance, I noted the most seriously involved eye on all records.

Table 29. Number of times that either the right or the left eye showed the more severe degree of Xerophthalmia.

	Boys		Girls		Together	
Age in yrs.	R eye	L eye	R eye	L eye	R eye	L eye
— $^1/_2$ yr	5	1	—	3	5	4
— 1 yr	1	4	3	5	4	9
— $1^1/_2$ yr	4	7	5	2	9	9
— 2 yr	4	4	5	4	9	8
— $2^1/_2$ yr	5	4	10	5	15	9
— 3 yr	—	3	4	4	4	7
— $3^1/_2$ yr	6	3	4	3	10	6
— 4 yr	1	1	1	—	2	1
— 5 yr	2	2	—	2	2	4
— 6 yr	—	1	1	1	1	2
— 7 yr	1	—	—	—	1	—
Total under 2 years	19	20	23	19	42	39
Total over $2^1/_2$ years	10	10	10	10	20	20
Total all ages	29	30	33	29	62	59

Conclusion : From table 29 there cannot be found a distinct difference in severity of Xerophthalmia in the right and the left eye ; from these data it cannot be decided that trauma has any influence.

Bacterial flora of the conjunctiva in eyes with a **different degree of Xerophthalmia.** There were 59 boys and 62 girls in whom there was found a different degree of severity of Xerophthalmia in the two eyes. In 38 boys and 44 girls a conjunctival smear of each eye was made, stained with methyleneblue and examined. No cultures were made. In this too simple way we found smears free from bacteria, with the same type of flora in both eyes, and with a different type of flora in the two eyes. Results in table 30 show that in 66% of the cases in boys with different degree of Xerophthalmia also a different type of bacterial flora was encountered, and in 82 % of the girls.

Table 30.

Boys				Girls			
same	different	negative	total	same	different	negative	total
12	26	—	38	7	35	2	44

Conclusion : A different degree of severity of Xerophthalmia in the two eyes was in the majority of cases accompanied by a different type of bacterial flora. Which was the cause, which the effect, could not be determined by this preliminary type of examination.

RELATION BETWEEN XEROPHTHALMIA AND NUTRITIONAL STATUS.

As there are many parallels between Xerophthalmia, especially in its severe degree, and general malnutrition, several signs related to nutritional status were recorded for each Xerophthalmia patient. I tried to apply the malnutrition index of Oomen in all cases which were examined by the pediatrician dr. Gondosoebarijo, but had to leave out the point on "height" as the children were not measured. Of the 9 remaining points of the index I compared the point relating to the condition of the eyes to the sum of the other points (rest-index). The index shows a lower figure than that in other publications, as the point of "height" is left out of the addition. I analyzed in this way 216 boys and 141 girls, seen between 1/8' 65 and 1/8'66. Of them 38 boys and 38 girls had been admitted to hospital as their general condition made this necessary, either in the Eye hospital Undaan, or in the Research Institute. I calculated the average index for each degree of Xerophthalmia, for boys and girls separately

Table 31. Comparison between average malnutrition index and degree of Xerophthalmia.

	Boys				Girls			
	X0	X1	X2	X3	X0	X1	X2	X3
Number of patients	14	121	19	62	8	62	20	51
average index	1.3	3.6	6.6	7.4	3.4	5.7	6	9

There is a distinct parallelism visible between the degree of Xerophthalmia and the general nutritional status as shown in the malnutrition index. Of the children who were admitted to hospital this could not be said, as we naturally admitted children who were in a bad condition. There are a number of children where the degree of Xerophthalmia thus is not parallel with the general nutritional status, but where the latter is worse. One of the explanations could be, that these children did grow very slowly or not at all, and did not need use up all of their vitamin A-stores for that reason (compare McLaren'sf indings in rats, 256, p. 111).

Table 31 a. Comparison of degree of Xerophthalmia and general malnutrition in Xerophthlmia patients admitted to hospital.

	Boys			Girls		
	X1	X2	X3	X1	X2	X3
Number of patients	5	7	26	8	4	26
average index	11	7	8.2	11	$11^1/_2$	$9^1/_2$

Generally the nutritional status of girls was already worse at the first eamination than that of boys. It seems that boys in spite of a better general nutritional condition are more sensitive to a deficiency of vitamin A than girls, showing clinical signs earlier. In agreement with their general condition, there were more deaths among our girl patients than among the boys. (see also chapter 26).

Criteria used in the determination of the malnutrition-index (according to Oomen, 346, 1955).

Judged as :	1	2	3
1. Insufficient Weight	Weight between 70-80% of standard	Weight between 60-70% of standard	Weight between 50-60% of standard.
2. Insufficient Height	Height between 85-90% of standard	Height between 80-85% of standard	Height between 75-80% of standard
3. Atrophy of subcutaneous tissue	Subcutaneous fatty tissue slightly shrinken	Clear emaciation	Extreme emaciation.
4. Oedema	Limited to the foot	Limited to the region of the leg below the knee	More extensive than 2
5. Depigmentation of hair and skin	Dubious depigmen-tation	Slight to moderate (mostly limited to face, chest and hair)	Clear depigmentation and hair-alterations

6. Xeratrophia cutis	Dubious alterations	The skin is dry, wrinkly, scaly, glossy; ther is a decrease in sweat & sebum excretion	Severe atrophy of the skin, sometimes small erosions or exfoliations
7. Crazy pavement dermatosis	Only in a small area	Limited to an area less than 9% of the surface of the body	An extensiveness more than 9% of the bodysurface
8. Muscle atrophy	Slight atrophy	Moderate atrophy	Extreme atrophy, mostly the children are not able to stand
9. Xerophthalmia	Xerosis conjuncti vae with or without Bitot spots	Xerosis corneae with or without ulcus corneae	Blindness by Xerophthalmia (destructed corneae)
10. Enlargement of the liver (measured in right nippleline)	Less than 2 cm below the costal margin	Less than 4 cm below the costal margin	More than 4 cm below the costal margin

As a standard of weight I used the Table from Tan Eng Dong, Soekonto and de Haas (501).

PLACE OF RESIDENCE.

In 1966 the percentage of patients living outside Surabaja was as follows :

Table 32. Relation of place of dwelling and degree of Xerophthalmia.
In 1966 lived outside Surabaja the following percentages of patients :

	X0	X1	X2	X3	X4
Boys	11%	7%	13%	59%	40%
Girls	15%	7%	33%	40%	80%
Together	13%	7%	23%	50%	60%

The children brought from outside the city were often in a worse condition than those from the city itself, possibly because they had been ill for longer living at a greater distance from competent help.

55% of the total number of boys suffering from severe Xerophthalmia came from outside the city, 50% of girls with X2,3,4.

DURATION OF ILLNESS BEFORE FIRST EXAMINATION.

In an effort to get an impression how quickly different degrees of Xerophthalmia develop, I asked how long the eye symptoms had been observed before the first examination. As always the history is not entirely reliable, e.g. there were very few children who had been showing eye disease between 3 and 4 weeks of time. Another way of collecting these data does not exist, so I give the results with reservations.

Table 33. Duration of eye symptoms before the first examination. Number of cases which stated a certain period of duration.

Duration	Boys				Girls			
	X0	X1	X3		X0	X1	X3	
— 7 days	12	56	28	96	6	33	26	65
7—15 days	7	72	34	113	3	37	26	66
15—22 days	3	43	17	63	2	15	19	36
22—30 days	—	—	—	—	—	—	2	2
1— 2 month	1	24	14	29	3	17	14	34
2— 3 months	1	3	7	11	—	6	7	13
3— 4 months	—	7	2	9	—	—	4	4
5— 6 months	—	—	1	1	—	—	—	—
1 year	—	2	—	2	—	1	—	1
no data	4	18	17	39	2	10	8	20
Total	24	207	103	363	14	109	98	241

Table 33 a. Percentage of each degree of Xerophthalmia which had existed for a certain period.

— 7 days	50%	27%	27%		42%	30%	26%	
7—15 days	29%	35%	32%		21%	34%	26%	
15—22 days	12%	20%	16%		14%	13%	19%	
22—30 days	—	—	—		—	—	2%	
1— 2 month	4%	13%	13%		21%	15%	14%	
2— 3 months	4%	1%	6%		—	5%	7%	
3— 4 months	—	3%	2%		—	—	4%	
5— 6 months	—	—	1%		—	—	—	
1 year	—	1%	—	—	—	1%	—	

Note: In Table 33 and 33a the cases where data on the duration were lacking are left out in the calculation of the total.

Conclusions : Nightblindness seems to many parents to be alarming enough to bring their children to the doctor, but as the only symptom it seems to be rarely to exist for more than one month.

Severe Xerophthalmia is in 25% of cases not older than one week and in 50% of cases not longer in existence than two weeks.

Especially among the girls there are rather many who have shown signs of Xerophthalmia for a month or more before they are brought for treatment.

From the figures for Xo and X1 it can be concluded that they can be in existence for rather long periods without increasing in severity. Probably they only progress to X3 if there is another causal factor added to the vitamin A-deficiency itself (either severe malnutrition, or an infection).

Response of nightblindness to treatment with vitamin A.

Of 200 boys with Xo, 1 at least 170 were nightblind, of 99 girls 91.

Table 34. Of nightblind children after treatment the nightblindness responded as follows:

Not nightblind after	4 days:	6 boys,	4 girls; still nightblind:	9 boys,	5 girls
	7	20	14	2	4
	14	11	4	—	—
	25	—	—	1	—
	8 months	—	—	1	—

On the rest of the cases there are no data, either because the patients did not return, or by my oversight.

Routine treatment in outpatients with Xerophthalmia consisted in injections of 50.000 IU vitamin A, cod liver oil and in many cases extra protein food supplements. If children were admitted to hospital their diet was of course devised with an eye to combating their malnutrition, and treatment for other diseases, infections and infestations given as necessary.

SERUM VALUES OF VITAMIN A, CAROTENE AND PROTEIN IN CHILDREN WITH XEROPHTHALMIA

admitted to Research Institute between 1/8 '65 and 1/8 '66.

Table 35. By degree of Xerophthalmia and sex.

No.	X	Sex.	Age	Vit. A	Carot.	Tot. Prot.	A/G ratio	Eye	Index
BOYS.									
49	X1	m	$1^{1}/_{2}$ yr	38 — 56	8 — 97	7.2 — 7.3	1.1 — 0.97	N	12
87	X1	m	16 m	23 — 30	17 — 40	6.7 — 5.6	0.8 — 0.94	N	12
472	X1	m	$3^{1}/_{2}$ yr	13 — 15	23 — 35	4.8 — 7.3	1.02 — 1.2	N	11
169	X2	m	2 yr	13 — 34	?? — 6	5.2 — 7.3	0.77 — 2.1	N	11
171	X1X3	m	$1^{1}/_{2}$ yr	28 — 30	29 — 8.3	6.7 — 7.8	0.46 — 2.2	O	11
514	X1X2	m	11 m	22 — 24	18 — 43	6.7 — 6.9	0.98 — 1.1	N	10
550	X1X3	m	16 m	24 — 35	15 — 70	7.1 — 7.1	0.79 — 1.3	O	8
572	X3X1	m	3 yr	25 — 37	18 — 76	7.0 — 7.6	1.2 — 1.1	ON	9
346	X2X3	m	$2^{1}/_{2}$ yr	14 — 16	12 — 12	8.3 — ?	0.9 — ?	N	2
54	X3	m	$2^{1}/_{2}$ yr	35 — 31	39 — 138	? — 6.8	? — 2.7	N	4
99	X3	m	3 m	26 — 25	18 — 44	7.2 — ?	1.3 — ?	OO	8
221	X3	m	$3^{1}/_{2}$ yr	24 — 14	? — 29	7.5 — 7.7	0.38 — 0.8	N	10
391	X3	m	$1^{1}/_{2}$ yr	29 — 23	16 — 33	7 — 6.8	1.2 — 1.03	OO	8
486	X3	m	3 yr	15 — 19	6 — 25	5.5 — 6.3	0.9 — 1.1	N	11
322	X3	m	$1^{1}/_{2}$ yr	25 — 37	43 — 38	8.4 — ?	0.7 — ?	OO	9
GIRLS.									
50	X1	f	4 m	18 — 29	22 — 53	5.4 — 7.1	1.2 — 1.6	N	7
82	X1	f	3 yr	41 — 26	38 — 39	5.8 — 7.3	1.14 — 1.0	N	15
86	X1	f	9 m	4 — 28	7.4 — 11	5.8 — 6.8	0.63 — 1.2	N	10
161	X1	f	3 yr	11 — 25	17 — ?	8.4 — 7.6	1,0 — 1.28	N	9
249	X1	f	3 yr	26 — 30	47 — 34	6.8 — ?	0.7 — ?	N	6
315	X2X1	f	3 yr	14 — 14	12 — 17	7 — 8.4	0.86 — 1.1	N	16
2	X3X1	f	13 m	30 — 23	17 — 44	6.9 — 7.2	0.9 — 1.2	N	4
75	X1X3	f	4 yr	52 — 27	63 — 12	6.1 — 6.3	0.56 — 0.97	NO	6
216	X1X3	f	7 m	12 — 14	11 — 13	7.2 — 7.2	0.6 — 1.3	O	9
189	X2X3	f	$2^{1}/_{2}$ yr	15 — 20	34 — 18	7.2 — 7.8	1.03 — 0.86	N	11
259	X2X3	f	2 yr	27 — 29	30 — 19	8.1 — 8.1	0.58 — 0.55	N	10
317	X3X2	f	2 yr	29 — 31	19 — 37	8.1 — 4.9	0.5 — 0.88	O	16
74	X3	f	2 yr	27 — 29	13 — 53	7.2 — 7.9	0.54 — 1.15	OO	12
433	X3	f	6 m	22 — 19	30 — 20	6.7 — 6.0	1.2 — 1.3	O	3
457	X3	f	$2^{1}/_{2}$ yr	44 — 36	32 — 43	6.4 — 7.3	2.06 — 1.2	OO	7
14	X4	f	3 yr	18 — 21	52 — 47	7 — 6.9	0.9 — 1.2	OO	3

Table 36. Percentages of total cases of various degrees of Xerophthalmia in which serum levels rose after treatment.

serum level of:	X1	X2	one eye X3	both eyes X3	all X3 together
Carotene	87.5%	50%	50%	50%	52%
Vitamin A	75 %	50%	72%	25%	52%
A/G ration	62.5%	100%	72%	75%	73%

Note : In Table 35 in each column the first figure is the level on admission, the second one after 2-3 weeks of treatment. Condition of the eye is marked : N : not blind ; ⌀ : one eye blind ; ⌀ ⌀ : both eyes blind.

Comment on Tables 35 and 36 : the number of estimations which could be done was low, and the values found were also rather low, but in accordance with figures in many other publications. As there are indications that the vitamin A-level rises with age, averages for differe at ages were calculated.

Table 37. Serum levels of Xerophthalmia patients, admitted to the Research Institute between 1/8'65 and 1/8'66, before and after treatment.

Age group	Carotene		Vitamin A		A/G ratio	
	before	after	before	after	before	after treatment
0—1 number of pat. average value	7 19.3	7 31	6 17.3	7 22	7 0.95	6 1.23
1—2 yrs number of pat. average value	8 19	8 42.2	8 27	8 31	8 0.87	6 1.27
2—5 yrs number of pat. average value	17 26.5	19 36	19 23.6	20 24	20 0.75	19 1.2

From a number of children only once there was made an estimation of the serum levels: if these are added, the following average values result :

Table 37a.

Age group	Carotene		Vitamin A		A/G ration	
	before	after	before	after	before	after treatment
0—1 yr number of pat. average value	9 15.5	10 26.5	8 19.5	10 20.2	11 1.06	8 1.14
—2 yrs number of pat. average value	10 18	10 35.8	10 26.4	9 29.5	10 0.92	7 1.27
2—5 yrs number of pat. average value	20 26	22 32.7	22 22.7	23 23.4	25 0.88	1.2 1.2

de Haas et al. (1940, 162) gave the following values :

Age	Number	Carotene Kerophthalmia	vitaminA	number	Carotene „normal”	vitamin A
0—1 yr	47	18.3	8.3	36	26	12.2
1—2	16	18.7	7.0	13	46.8	26.9
2—5	36	22.5	7.0	12	69.7	19.8

The Surabaja figures show a higher vitamin A-level than those formerly found at Djakarta in Xerophthalmia cases, even higher than the so-called normal figures in Djakarta children. This could be the result of a different method of estimation, but Carotene values are in agreement in both groups.

Conclusion : Xerophthalmia still is the most important eye disease in young children. The frequency is continuously rising, and the percentage of infants with severe (blinding) Xerophthalmia is increasing. These latter cases need prompt hospital treatment, by which it is sometime possible to save part of the eyesight in one or both eyes. Facilities for hospital treatment of these patients at Surabaja are insufficient from lack of room in the existing children's wards. Even with the best treatment it is not possible to save the life and normal eyesight of more than 50% of Keratomalacia-patients. Maximum interest in this field is necessary in view of these facts.

Chapter Twenty six.

INVESTIGATION INTO THE FATE OF SMALL BLIND CHILDREN IN EAST JAVA.

(Made possible by a grant from the National Research Council of Indonesia).

Teamleader : Prof. M. Soetopo. Investigator : J. ten Doesschate.

Purpose of the investigation :

The fate of many small children who are seen early in life by the ophthalmologist and who are blind, is uncertain. It is important to know how many of those children survive and are eventually in need of adequate schooling and/or vocational training. Also from a medical point or view it is desirable to know if improvements in treatment or prevention can be worked out.

Material of the investigation :

During the years 1958 through 1962 all blind persons who are examined by the outpatients' dept. of the Eye hospital Undaan at Sourabaya were registered. At the same time the vision if any, and the cause of the blindness were noted, if the latter could still be ascertained. Not only recently blind persons, but also the patients who had been blind for a long time were registered.

From this list the children who were less than 16 years of age at the time of the registration were divided into those living in the city of Sourabaya and those living outside, mostly in East Java.

There were 250 children stated as living in Sourabaya and 459 children living outside Sourabaya.

For practical purposes only the children with an address in the city of Sourabaya were visited at this time.

Method of investigation :

During the months of September through December 1964 a trained social worker was provided with lists of addresses of blind children presumably living in Sourabaya, and did home visits with the purpose of determining :

1. whether the children were alive,
2. whether they went to school,
3. whether they had been moved to other addresses,
4. how the conditions in the homes were, hygiene, economy, composition of the family etc.

Comment :

It was known that a number of the children, who were stated as living at a Sourabaya address, were only brought in from their villages for the eye examination. Partly because their parents do not think it important to explain the real situtation, and partly from fear that out of town patients will not be treated the statements can not always be trusted.

Quite a large number of children who go blind early in life, is suffering from keratomalacia, and it is suspected that these children have a shorter life-expectancy than normal children.

It was also known that some of the children whose eyes looked blind for life, did recover some vision afterwards, contrary to expectations.

The social worker was able to see for herself how difficult the various homes were to be reached, and she used the opportunity to advise the parents on the early training of blind children.

She tried to induce them to bring their children to the Eye Hospital Undaan for a reexamination.

As the original examinations were done during 1858-1962 the time lapse was at least $1^{1}/_{2}$ and at most $6^{1}/_{2}$ years.

Results of investigations by home visits, completed with reexaminations of the blind children if possible, can be described under the headings as seen in Table a.

Table a.

Fate of blind children registered during 1958-1962 as living at Sourabaya.

Dead	83	
Probably dead	5	
Living outside Sourabaya	15	(new address known)
Living ouside Sourabaya	22	(no address known)
Moved inside the city	1	(new address known)
Moved inside the city	2	(no address known)
No trace to be found	30	(
Not blind any more	13	(reexamined)
Not blind any more	13	(not reexamined)
Still blind	5	(not reexamined)
Still blind	11	(reexamined : 4 can be operated)
Still blind	10	(sent to school for the blind, JPAB).
Still blind	24	(already pupils of JPAB).
Not visited	16	
Total	250	(40 not visited)

Comment :

Dead : From 6 children it was known that they had died shortly after registration.

From 9 children it was known that the general condition was bad at the time of registration.

11 children had been brought to a village and died there. Probably dead : relatives or neighbours told the social worker at the time of the home visit that the child was said to be dead.

Living outside Sourabaya with a known address : the people living at the registration address were relatives and knew that the blind child was still alive. The group with unknown address outside town contains children of whom generally is known to which town they went, but no exact address. The children are supposed to be still alive.

No trace to be found : at the address given they were unknown, also at the nearest official centre (rukun kampung, kepala kampung).

This is possibly caused by the lapse of time, especially as the older registration years contained most of these cases :

1958 : 9
1959 : 11
1960 : 1
1961 : 6
1962 : 6

Also it is to be noted that the dwellings are often temporary in the first place (gubug), and that many changes take place in the parts of the town concerned : whole streets are changing names, numerotation, or are even disappearing completely, as the temporary dwellings are demolished to make room for larger buildings.

It is probable that from the 30 children of whom no trace was to be found the same percentage has died as from others, i.e. 88 from 220, ca 40%, so from the 30 probably 12. From 2 it was already noted at the time of registration that their general condition was bad. The percentage of children suffering from keratomalacia was circa the same as in the whole group (compare table b and c).

Not blind any more : There were 26 children who had recovered a certain amount of eye sight. Many of them were going to normal schools ; those who went to schools out of town could not be induced to be reexamined. 13 were seen again and of them : 3 had an amount of vision which belongs in the group "blind" (1/60 or less), but went to normal schools. 4 had 3/10 in the best eye, 6 were monoculus. 2 of them are pupils of the school for the blind JPAB.

It seems that the normal schools to which a number of these children with a far too low visual acuity went, made no objections as long as the children are clever enough to absorb all the information they need with some extra help from their comrades, permission to go near the blackboard etc. It remains strange that none of the educators ever asked for any kind of advice from the eye doctor, and also that schooldoctors or schoolnurses never referred these children back to the Eye Hospital for a check-up.

Still blind : Some of the children who were reexamined suffered from cataracts which can be removed.

It was not necessary to visit the old pupils of the JPAB ; among the children who came for a check-up after the home visit there were 10 who were referred to the school.

For 16 children it was not deemed necessary to visit them, because they were recently seen, or because they had become over 16 years of age meanwhile, and so were not to be counted as "children" any more.

8 children were blind as before, but were either still too small for the school, or their parents were not willing to send them, often because of transportation difficulties.

Conclusions :

1. From a total number of children of 250 registered as living at Sourabaya there had disappeared from view after a few years 30 12%

From the rest	88	had died i.e. 40% of the rest	
	37	were living outside the city	17%
	26	recovered some sight	12%
	35	were blind, went to the blindschool	15%
	35	were blind without adequate schooling	15%

Of the blind children who can be supposed to be still alive ca. 340 do not get adequate schooling, if we add to the 90 covered in this investigations, the interpolated figure for the children registered as living outside Sourabaja :

> total 460, still alive probably 276, (of whom ca 25 may have recovered some sight), all of whom live out of reach of the school for the blind at Sourabaya.

2. 40% of small blind children will probably die at an early age.

3. Blind children are often brought from the city to villages, possibly as it is easier to feed them there and to keep them out of harm from modern traffic etc.

4. Only 1 in 10 of small blind children recover some eyesight, and very little at that.

5. There should be more cooperation between educational authorities and medical ones, as it became evident that a few (and possibly far more) almost blind children were going to normal schools, often with great (and unnecessary) difficulties.

6. For adequate schooling of blind children it is essential that an efficient way of transportation to the school can be worked out.

7. For the many blind children dispersed over various small villages there should be worked out a way of either teaching them at home (itinerant teachers specially trained for this work), or putting them in boarding schools, as transportation difficulties otherwise will be so great, as to completely obstruct any kind of education.

Medical aspects of children's blindness in Sourabaja and East Java.

The first fact which is clear from table b and d (the division of children according to cause of blindness), is that there are many more blind boys than girls. Yet the number of boys and girls who were brought to the outpatients' dept. of the Eye Hospital during the registration years was evenly divided. For eyediseases in general there seems to be no tendency to give more attention to boys than to girls, but maybe in the case of blindness the parents think it more important to go to the utmost to get help in the case of a blind boy than in the case of a blind girl. This is a supposition which is not improbable, but for which there is no evidence. From the lists of causes of blindness it is evident though, that there are more boys suffering from keratomalacia (malnutrition) than there are girls, in Sourabaya there were registered 77 boys against 51 girls, outside the town 113 boys against 63 girls. The mortality rate which could only be ascertained for the children at Sourabaya is : 42% for the boys, against 47% for the girls. The results of this, if it could be generalized for the children living out of town also, would be that the difference between the numbers of living boys blind by malnutrition and girls by this cause, would be still greater. Again there is no evidence that this is a case of greater attention given to the blind boy, with the result that he will survive, whereas blind girls possibly are given less care and consequently die. Possibly also the boys have a better chance to survive an A-dificiency, which is generally not the case in experimental work on A-deficiency, where mostly the female does better on less A-vitamin.

It is to be noted, that part of the group "unknown" certainly also should be counted as from malnutrition, but as they were seen in a late phase (mostly leucoma adhaerens) it could not be ascertained if they were caused by malnutrition, gonorrhoea or some other cause (code numbers 98, 99).

Of the children who could not be traced there were even higher numbers registered as blind by malnutrition i.e. 55% of the boys, and 66% of the girls. As it is probable that these children are either dead or also moved to villages, a few of them will have to be added to the number of blind by malnutrition who are now living out of town (table c.).

From the children, who certainly were moved to villages and who are probably still alive, 55% were suffering from malnutrition blindness. This agrees with the overal percentage, so there seems no tendency to bring especially the sufferers-from malnutrition to the villages (table e).

Also from the children who recovered some eye sight the percentage for boys and girls was almost the same (table f). So this will not finally alter the difference in numbers of blind boys and blind girls.

The other cause of blindness are fairly evenly divided over boys and girls (table b and table d) and the numbers of dead, vanished, or seeing again, do not make any difference, between boys and girls (table g).

It remains probable from the present evidence that there really are more boys than girls suffering from keratomalacia, and that this difference accounts for the higher number of blind boys.

It is clear also, that high numbers of children who are blinded at an early age are actually living in the villages, and that this blindness is for a large part preventable, as it is caused by keratomalacia. Therefore it seems highly important to bring the knowledge of the diseases as far as possible to the periphery, especially to give it the highest attention at the points where there is contact between medical personnel and small children, i.e. the BKIA's. The advice given to the mothers should be centered on early addition of green vegetables to the children's food. Also in the case of treatment of all kinds of infections it should be known that fever in any form causes a loss of Vitamin A. which can quickly lead to total depletion. In treating any kind of respiratory infection, as well as in all kinds of diarrhoea, it is essential to give extra supplies of Vitamin A right from the start. This cannot be repeated often enough, as the sequelae of mal-treatment of these cases are still over frequent.

In circa 85% of our cases of Xerophthalmia there was a history of a recent infectious disease, measles, chickenpox, bronchitis, dysentery (also ascaris). Even if this disease was treated and cured, often Xerophthalmia developed yet, pressumably because the stores of Vitamin A, which usually are low in children in this country were quickly depleted by the fever (see : T. Moore, "Vitamin A", chapter 32, Elsevier 1957).

The Eye Hospital Undaan tried to do as much as possible for the children suffering from Xerophthalmia generally, also the lighter cases who were not yet blind by keratomalacia. The Eye Hospital got the help from a pediatrician who came once a week and saw all of the cases of eyedisease caused by any kind of general disease, malnutrition, tuberculosis, congenital malformations, congenital syphilis. As a further intensification of this work there was made an opportunity at the Research Hospital P4K to admit children who needed more specialised care, and quite a large number of children suffering from eyedisease caused by malnutrition were admitted from 1960 on. The results of this work can be shown in the figures for blindness caused by malnutrition which show a steadily decreasing tendency during the latter years (table h). In this table the numbers of children presented at the outpat.dept. of the Eye Hospital suffering from Xerophthalmia are shown also, and these figures are decreasing during the years, but not in the same degree as the figures for children finally blind by the disease. So we feel that it can be said that the results of early and thorough treatment of Xerophthalmia patients with full attention to the whole child, not only the eyes, is very rewarding. dr. Soerti Gondosoebaryo who undertook this work for our patients in often desparate looking cases, can show very satisfactory results, as it was often possible to at least restore the sight of one eye, if not both, by her specialized care.

Blindness not caused by malnutrition.

Next in importance as a cause of blindness, as well in the list of children registered in the town of Sourabaya (table b) as outside Sourabaya (table d) is the group of congenital and hereditary diseases or malformations. This group contains many congenital cataracts, part of which can be operated as far as there are no other malformations present (microphthalmus especially). In some cases the mother still remembered to have been feverish early in pregnancy, which made it probable that the cataracts of the child were caused by a viral infection of the mother. Very often the mother did not remember anything of a slight illness, as it was too long past and not thought important. Really hereditary diseases and malformations there certainly must be more than are shown, but in the present conditions of very unstable family relations, questioning into heredity mostly meets no clear response. Only in a few cases of congenital cataracts the disease was clearly familiar, and there was a family with retinoblastoma in several children, who in spite of this known tendency still brought the children in a far advanced stage.

As is known from advanced countries where prevention of blindness is nearly perfect, the hereditary causes of blindness are by far the most important, as they are the most difficult to prevent.

The next group in importance is the one caused by gonorrhoea. A part of this only is caused by infection at birth, quite a large number is caused by infection from eye to eye. The figures of blindness caused by this infection are too small to be able to see if there is a tendency for them to decrease during the years.

The group "unknown" must be divided over the most important causes : keratomalacia, gonorrhoea and "other causes", but there are no clear indications how they should be divided.

The group "neurological disorders" contains: hydrocephalus, encephalitis, meningitis (if by tuberculosis they are stated under that disease) and shows rather a high mortality rate as is to be expected (table g).

Medical conclusions :

1. By far the most important cause of blindness in Sourabaya and East Java in the age group under 16 years is : malnutrition with ensuing Keratomalacia (127 children from 250 blind ones living in Sourabaya and 173 from 459 living outside Sourabaya).
2. More boys than girls are blinded by keratomalacia.
3. Many of these children are living in villages.
4. Presumably the disease is caused by a combination of : the usual food which contains too little vitamin-A sources ; and intercurrent infections which act like a trigger mechanism causing the outbreak of Xerophthalmia.
5. It is therefore important to give full attention to : improvement of the usual food by propaganda at the BKIA's etc. for : early addition of green vegetables to babies' food; consumption by pregnant and lactating women of green vegetables and yellow fruit.
6. Attention of the doctors should be called to the necessity of supplying small children suffering from an infection with vitamin A by the parenteral route quite from the start of the treatment.
7. Far less important in this age group is the next "preventable" cause of blindness, i.e. gonoblennorrhoea (13 from 150 blind children in Sourabaja, 30 from 459 outside Sourabaya).
8. As circa 60% of the gonoblennorrhoea cases in children are **not** contracted at birth, but by contact infection from eye to eye, everybody concerned with mothers and children should help to teach them not to wipe the faces of the whole family with the same (dirty) slendang.
9. Part of the congenital blindness could be helped by operation, making it advisable for teachers etc. to send all children with visual handicaps to an ophthalmologist at least once.
10. Trachoma is not an important cause of blindness in this age group.

Table b.

Causes of blindness in 250 blind children in Sourabaya 1958 — 1962.

Diagnosis	Boys		Girls		Total
	number	%	number	%	
12 Gonorrhoea	6	4	7	7	13
13 Morbilli	—	—	1	1	1
18 Variola	—	—	1	1	1
19 Syphilis	2	1.3	3	3	5
20 Trachoma	2	1.3	—	—	2
21 Tuberculosis	—	—	2	2	2
65 Neurological disorders	7	4	4	4	11
67 Malnutrition	77	51	51	51	128
89 Congenital & hereditary	32	21	18	18	50
91 Unknown to science	—	—	2	2	2
98 Unknown Wr. negat.	4	2.6	1	1	5
99 Unknown	21	14	9	9	30
	151		99		250

Explanation : The figures in front of the diagnosis are the numbers used in the international code of causes of blindness as accepted at the ophthalmological congress at Vienna in 1964.

Table c.

Blind children who could not be traced by home visit, according to cause of blindness.

Diagnosis	Boys		Girls	
	number	%	number	%
20 Trachoma	1	5.5	—	—
21 Tuberculosis	—	—	1	8
67 Malnutrition	10	55	8	66
89 Congenital	3	16.6	1	8
98 Unknown Wr. neg.	1	5.5	—	—
99 Unknown	3	16,6	2	16
	18		12	

Explanation: percentages calculated from the total of boys or girls who could not be found.

Table d.

Blind children registered as living outside Sourabaya, according to year of registration and cause of blindness.

Boys.	1958	1959	1960	1961	1962	Total	%
12 gonorrhoea	1	5	2	4	3	15	5.7
13 morrbilli	—	—	—	—	—	—	—
18 variola	1	—	—	—	—	1	
19 syphilis	1	1+—	—	—	2	1+3	1.5
20 trachoma	3	1	1	—	1	6	2.3
21 tuberculosis	—	—	—	—	1	1	
22 typhoid fever	—	—	—	—	—	—	
29 infection in pregnancy	—	—	—	—	—	—	
43 trauma	—	—	—	—	1	1	
65 neurological disorders	1+ 1	2+ 1	3	2+1	1+3	9+ 6	5.7
67 malnutrition	1+20+4	1+29+2	24+3	16+3	1+7+2	3+96+14	43
81 hereditary		1+—	—		—	1+	
89 congenital	4	6	5	9	12	36	13
91 unknown to science	—	1	—	1	—	2	
98 unknown Wr. neg.	4	2	1	3	3	13	5
99 unknown	16	10	10	8	6	50	19
						5+233+20	

Girls.							
12 gonorrhoea	5	5	2	2	1	15	7.5
13 morbilli	1	1	—	—	1	3	1.5
18 variola	1	1	—	—	1	3	1.5
19 Syphilis	—	—	3	2	—	5	2.5
20 Trachoma	—	—	—	2	—	2	1
21 tuberculosis	—	—	—	—	—	—	—
22 typhoid fever	—	—	1	—	—	1	
29 infection in pregnancy	—	—	—	—	—	—	
43 trauma	—	—	—	—	—	—	
65 neurological disorders	2	2+ 1	—	2	1	7+ 1	4
67 malnutrition	1+9+2	1+20+1	3+5	10+2	9	5+53+5	31
81 hereditary	—	—	—	2	—	2	1
89 congenital	3	6	8	6	13+1	36+1	18
91 unknown to science	1	1+—	—	—	2+1	3+2	2.5
98 unknown Wr. neg.	2	1	3	2	—	8	4
99 unknown	16	9	7	1	6	48	24
						8+186+7	

Explanation: if more than one figure are stated, the **one** is the central one, the figure in front of it is the number of children who are dead, the figure after the **one** is possibly dead, as the general condition at the time of registration was bad.

Table e.

Blind children who were brought to villages according to cause of blindness.

Diagnosis	boys	%	girls	%	Percentage of total
12 gonorrhoea	5	83	2	28	17.5
21 tuberculosis	—	—	1	50	2.5
65 neurological disorders	—	—	2	50	5
67 malnutrition	15	20	7	14	55
89 congenital	1	3	2	11	7.5
99 unknown	3	12	2	20	12.5
total	24		16		100 %

The % under boys and girls are calculated from the total of this diagnosis. The last column gives the percentage of the total of children who had been brought to villlages.

Table f.

Children registered as blind who recovered some eyesight.

According to cause of blindness.

	Boys	%	Girls	%
12 gonorrhoea	1		—	
20 trachoma	1		—	
65 neurological disorders	—		1	
67 malnutrition	6	35	3	33
89 congenital	5	33	3	33
99 unknown	3	20	3	30
total	16		10	

Explanation : % calculated from total number of children in this table (boys or girls).

Table g.

Fate of 250 blind children as found by home visits divided according to diagnosis.

Diagnosis	Boys					Girls				
	Dead	%	seeing	%	blind	Dead	%	seeing	%	blind
12 gonorrhoea	1	16.6	1	16.6	4	2	28	—	—	5
13 morbilli	—	—	—	—	—	—	—	—	—	1
18 variola	—	—	—	—	—	—	—	—	—	1
19 syphilis	1	50	—	—	1	1	33	—	—	2
20 trachoma	—	—	1	50	1	—	—	—	—	—
21 tuberculosis	—	—	—	—	—	—	—	—	—	2
65 neurological disorders	3	42	1	14	3	1	25	1	25	2
67 malnutrition	32	42	6	7.8	39	24	47	3	5.8	24
89 congenital & hereditary	12	37	5	15	15	5	27	3	16.6	10
91 unknown to science	—	—	—	—	—	1	50	—	—	1
98 unknown Wr. negat.	—	—	—	—	4	—	—	—	—	1
99 unknown	5	23	3	14	13	—	—	2	22	7
	54		17		80	34		9		56

Explanation : the percentages are calculated from the total of the boys or girls with the same diagnosis.

Table h.

Number of deaths of children blind by malnutrition according to year of registration.

year	boys	girls	boys	girls	boys	girls	Xerophthalmia cases
	blind from malnutrition		died		living		
1958	21	15	13	11	8	4	419/3244
1959	24	12	8	6	16	6	561/3721
1960	15	7	7	2	6	5	435/3717
1961	13	10	3	3	10	7	322/4673
1962	6	7	12	2	5	5	310/5357

Explanation : the figures for 'Xerophthalmia cases" are the total number of cases of this disease seen in the outpatients' dept. during the year on a total of children seen.

From Lembaga Kesehatan Nasional (formerly P4K) di Surabaja.

FOLLOW-UP OF CHILDREN WHO WERE ADMITTED TO P4K SOURABAYA WITH EYE DISEASE.

During the years it had become apparent in the Eye Hospital Undaan, that many small children who were brought for treatment were suffering from eye conditions which were caused by some more general disease, e.g. malnutrition and/or tuberculosis. The Eye Hospital itself was not able to give adequate care to these children, and was lucky in finding an opportunity to cooperate with the children's department of P4K; after having been seen by the pediatrician the children were admitted there and regularly seen by one of the eye doctors. Afterwards the parents were asked to bring their children for periodic check-ups to the Eye Hospital again at the time the pediatrician was there.

This cooperation started in 1960, and was carried on until 1966.

During the years it became clear that the children were not always brought for their check-up ; so as a social worker was temporarily available for work among blind children, she was asked to visit the children also, who had at one time been admitted to P4K and who had not shown up for their check-up, although presumably living at Sourabaya. The social worker could arrange for these visits easily along with those to the blind children as they were living in the same parts of the city. She visited a total of 53 ex-patients of P4K during the month of Nov. 1964. Results are shown below. All of the children were suffering from Xerophthalmia except one whose twin brother was admitted with this condition, but who was himself healthy as far as his eyes were concerned. There were 10 blind children among them, who were not covered by the register of blind children as they came after 1962.

Of 43 seeing children 15 were one eyed, the other eye being lost by Xerophthalmia. So from a total of 106 eyes 36 were lost by this disease.

From the seeing children 11 had been brought to a village ; 2 of them were brought for a reexamination.

4 changed their address inside the city of whom 2 came for reexamination.

25 were still living at the same place, of whom 23 were reexamined.

2 had died after having been brought to a village, one from smallpox.

From the blind children 2 were brought to a village,

1 moved to another address in town and was reexamined.

4 had disappeared without anybody at the given address knowing where they were.

1 had died after having been brought to a village.

2 were known to be alive, of whom 1 was seen again.

The purpose of these visits was sufficiently carried out in that 29 of 53 children were induced to come for a check up, so there is a chance that a deterioration in the general condition or in the condition of the eyes can be treated in time.

The numbers investigated are too small for definite conclusions, but it seems striking that among these children as among the children from the blind registration, there is the tendency for them to be brought outside the city as they are blind ; from the seeing children who were still alive (41) 29 were still living in town, whereas from 9 blind children who were still living 3 were still in town.

The social level from which most of the children come, contains many people who are not real townspeople, often they go back and forth to their villages, coming to town only for work. It seems natural that blind children in a town are more of a burden than in the village, and also are less safe from modern traffic and its dangers. The eventual educational possibilities of blind children do not yet play a part in the decision of their parents to let them live in town or in the village.

Chapter Twenty seven.

POSSIBILITIES FOR KERATOPLASTY.

On Java, and probably in other parts of Indonesia as well, the frequency of corneal causes of blindness is very high as compared to western countries : According to Sorsby (1966, 472) corneal disease in general caused 2.3% of the cases of blindness registered in 1955-1960 in England, whereas from the Tables for Undaan, Surabaja, 1958-1962, it can be seen that 51.1% of the cases of blindness were caused by corneal disease. A certain "spontaneous" improvement is going on, as the percentage for 1966/67 was only 42.1%, but a large residue of corneal blindness remains. It is therefore natural to look for methods to relieve this situation. One of the possibilities which come to mind (apart from prevention!) is keratoplasty, i.e. the transplantation of human cornea, usually taken from persons who have just died. Techniques for storage of donor material and for the best methods for surgery have generally been worked out to satisfaction, but there seems until now not to have emerged a general concensus as to indications and contraindications for this type of surgery.

We are here only concerned with the methods of keratoplasty which aim at visual improvement, as opposed to therapeutic or tectonic purposes. For the latter indications the field would be large enough, but the scarcity of donor material is prohibitive.

The best optical improvement is to be expected from penetrating forms of keratoplasty (Castroviejo, 50 p. 1858; Puchkovskaya, 383 p. 185) In these cases the whole thickness of the diseased cornea is removed and replaced with a whole thickness transplant from a healthy eye. But this operation also carries a greater risk than a lamellar keratoplasty, where only the diseased layers of the cornea are replaced by an equally thick transplant from a healthy donor eye. The latter method is far more innocuous than the former, and can on many occasions give optical results which are anyway better than the previous condition. Combinations of both methods are advocated too, in that first the receiving corneal bed is improved by a lamellar transplant, followed by a smaller penetrating graft, which then has a better chance of success for restoring useful vision.

Results which can be expected: Stucchi (1967, 487) in Lausanne, Switzerland, performed 111 keratoplasties, the lamellar ones giving mediocre results (because of poor recipient corneas). The best results were reached in cases of familial corneal dystrophies, then followed keratoconus. Both indications are extremely rare in Indonesia. Overall results : 36.6% good (i.e. visual acuity more than 1/10) in 54 interventions ; 16.3% fair (i.e. same visual acuity as before), in 24 interventions ; 47.9% bad (i.e. visual acuity worse than before) in 69 interventions.

The factors which are to be considered are :

1. Limits of visual acuity. Generally it is not considered justified to perform a penetrating keratoplasty if the other eye has good vision (Paton, 358 p. 61). If the visual acuity of an eye is over 1/10, there is no indication for keratoplasty, as firstly the risk of the operation is not negligible, and secondly the hope for improvement is too uncertain (Arruga, cited by Paton 358 p. 61, Barrios 107 p. 437-8).
2. Condition of the cornea. Most authors consider it necessary that there is a rest of healthy cornea, which will border on the graft to ensure proper healing (Paton, 1. c.p. 59, Castroviejo, 1. c.p. 1857). Deep vascularization is considered unfavorable, and should be treated previously, if possible several months beforehand, with corticosteroids, beta radiation, or a lamellar procedure, or a combination of these (Paton, 1. c.p. 65). Synechiae anteriores, according to most authors, have to be removed preliminarily, and glaucoma must be treated to ensure normal ocular tension during and after surgery. (Paton, 1. c.p. 60, Estrada, 437 p. 501). Several authors consider synechiae anteriores and glaucoma to be contraindications, which they certainly are in case they are intractable.

Fistula corneae : There are many difficulties in treating fistula corneae by keratoplasty : Löhlein (244) in his series saw improvement only in 2 of 15 cases. Often it only seems possible to effect tectonic improvement (Filatov, cited by Paton, 1. c.p. 87) and there often follows secondary glaucoma. It seems easier to reach tectonic improvement and to prevent the fatal late infection by the method used by Mulock Houwer (1941 313, 313a) using cyclodialysis. Kronfeld (216) seems to follow the same method, which also in my hands often results in closure of the fistula in at least 80% of cases. Kronfeld adds a keratoplasty afterwards.

Application in several clinical entities.

The causes of corneal disease leading to blindness which are most frequent here, i.e. Keratomalacia, gonoblennorrhoea and Trachoma, are still subject to conflicting opinions, but generally they do not belong to the favorable group.

1. **Xerophthalmia.** In this country blindness caused by Xerophthalmia mostly develops before the age of 3 years. This makes the prognosis for Keratoplasty unfavorable, for as Paton (1. c.p. 58) and Puchkovskaya (1. c.p. 77, 78 and 84) point out, the eyes of children who have gone blind at a very early age often are amblyopic. Paton quotes

Sourdille as saying, that Xerophthalmia in later stages is almost a total hindrance for performing a keratoplasty, but it is not clear if Xerophthalmia as the results of vitamin A-deficiency is meant, or a xerophthalmic process of the cornea as a result of chronic eye disease. Löhlein performed a keratoplasty in 12 cases where blindness by various causes had struck at a very early age, including keratomalacia, and saw success in only 2 of them.

2. **Conjunctivitis gonorrhoica.** Bietti (quoted by Paton, l. c.p. 79) considers eyes blinded by this cause as unfavorable (presumably ophthalmia neonatorum is meant with ensuing amblyopia). Löhlein had success only in 1 out of 7 cases and Barrios (l. c.p. 444) also considers them unsuitable for the procedure. The reason given is the same as that in Keratomalacia.

3. **Trachoma.** Keratoplasty in cases of corneal affections caused by Trachoma can be divided in 2 kinds, viz. cases where the procedure is applied therapeutically,, "a chaud", of which several authors claim to have seen satisfactory results (Wassermann, 549, 550), Giani and Foffano (139) Giani (140) and on the other hand in cases where it is used to improve vision after the disease has healed : Farnarnier et al. (109) saw improvement in 2 out of 5 cases. Barrios (l. c.) warns against the danger inherent in vascularization, and against a possible flare-up of the disease as the result of surgery. Puchkovskaya never saw a good result after subtotal penetrating keratoplasty in cases of Trachoma. Paton (l. c.p. 79) performed a keratoplasty in 11 cases : 4 of them penetrating, and one lamellar one had a good result, the others remained as before even after secondary penetrating procedures.

Possibilities of application of keratoplasty in East Java.

At the time of the investigation into the causes of blindness, 1958-1962, keratoplasty was still a very remote possibility at Surabaja. Since then the outlook has improved, and now it seems worthwhile to try and get an impression of how many blind people who come to the Undaan Eye Hospital could possibly, after all other ways and means have been exhausted, be helped by keratoplasty.

As a sample I collected the records of all patients seen by myself at the Outpatients' Department during the first half of the year 1965. With this idea in mind right from the start, the following data were recorded in each case: age, sex, visual acuity of each eye, cause of impairment of vision if any, outlook for improvement either by other forms of treatment or by keratoplasty.

The patients with visual impairment are divided into 2 groups : the group with lowered visual acuity in one eye only, which is not eligible for keratoplasty, and the group with lowered visual acuity in both eyes. Of the latter group a certain number were suffering from cataract, which could be helped by surgery either at once or eventually at a later time. There was a number of patients whose visual acuity improved during treatment, until visual acuity in one or both eyes rose above the limit of 4/60. Finally there was a rather large group where visual acuity was not lowered by corneal alffections, but by some other cause. This group naturally could not be candidates for keratoplasty.

Material.

During the first 6 months of 1965 I saw 3714 patients who came for the first time to the Outpatients' department. Cases who had been seen before were excluded to prevent overlapping with the 1958-1962 group. 2958 patients had no visual impairment at theat time lower than 4/60 in either eye. The definition in the 1965 group intentionally uses a different limit of visual acuity from that used in the 1958-1962 group, because I wished to consider all patients who in a real sense could benefit from keratoplasty: therefore patients with visual acuity between 1/60 and 4/60 had to be taken into account also. In other respects both groups of patients, that of 1958-1962 and that of 1965, are comparable : age groups, race, sex, social stratum do not differ in both groups.

Visual acuity.

From the sample seen during the first half of the year 1965 ca. 9% had visual acuity of less than 4/60 in both eyes, ca. 11% in one eye only, whereas ca. 79% had visual acuity of better than 4/60 in both eyes.

Table 38 **Total number of patients considered in sample 1/1'65—1/7'65.**

Visual acuity.	0—17 yrs	16—41 yrs.	Over 40 yrs. MEN.
4/60 & over	393	1030	411
1 eye less than 4/60	21	93	99
2 eyes less than 4/60	25	40	95
Idem, **WOMEN**			
4/60 & over	351	592	181
1 eye less than 4/60	43	76	78
2 eyes less than 4/60	23	41	122

Age and sex. The usual distribution in the material of the Outpatients' Department is as follows : under 17 years, 51% male, 49% female.
16-41 yrs. 62% male, 38% female.
over 40 yrs. 64% male, 36% female.

The surplus of women who are blind compared to the normal distribution is probably caused by the high number of women who go blind by glaucoma and complicated Trachoma.

Non-candidates for keratoplasty.

Apart from the whole number of monocular blind, there were other cases who had to be discarded as possible candidates for keratoplasty :

A. Patients, who improved so far, that the visual acuity in one eye rose above the 4/60 limit.

B. Patients, who improved so far, that both eyes had visual acuity better than 4/60.

C. Patients suffering from senile cataract, who could be helped by surgery. From 346 patients who had visual acuity of less than 4/60 in both eyes at first examination, the following numbers could be discarded for keratoplasty, as they belonged to groups A, B and C.

Table 39 **Non-candidates for keratoplassty, with visual acuity less than 4/60.**

MEN	0—17 yrs	16—41 yrs	Over 40 yrs
A. One eye improved	1	2	8
B. 2 eyes ,,	1	3	9
C. Senile cataract	—	—	21
WOMEN.			
A. One eye improved	—	3	12
B. 2 eyes ,,	—	3	2
C. (Senile) cataract	1	—	19

In the 1958-1962 group the categories A, B and C were not included, as they are not to be considered as permanent blind.

Classification according to the causes of blindness.

Using the International Standard Classification for Causes of Blindness, the remainder of the sample 1965 who were not discarded for one of the reasons already enumerated, were tabulated, according to sex and age groups, in the following six Tables. Cataract cases who still figure in the Tables are those, where surgery was refused, where it did not succeed in improving visual acuity over 4/60, or where there was a contraindication against surgery. Note: The visual acuity in the next Tables was less than 4/60, in the 1958-1962 Tables it was less than $1^1/_2/60$.

Table 40. 23 blind boys, under 17 yrs. of age, Undaan 1/1—1/7'65, visual acuity less than 4/60.

	13	18	21	65	67	89	99	Total
144						1		1
147						2		2
313		1	1					2
315					3		1	4
360	1				10			11
380.5					1			1
410						1		1
560				1				1
Total	1	1	1	1	14	4	1	23
%	4.3	4.3	4.3	4.3	69	17	4.3	100%

Table 41. 22 blind girls, under 17 yrs. of age, Undaan 1/1—1/7'65, visual acuity less than 4/60.

	12	13	18	21	65	67	81	98	99	Total
143							1			1
146							1			1
147							1			1
150	1		1			1				3
313	1		1						1	3
315		1				1				2
360						7				7
410							1			1
560					1			1		2
710				1						1
Total	2	1	2	1	1	9	4	1	1	22
%	9	4.5	9	4.5	4.5	41	18	1	1	100%

Table 42. 35 blind men, 16—41 yrs. of age, Undaan, 1/1—1/7'65. Visual acuity less than 14/60.

	12	18	19	30	28	40	89	91	98	99	Total
110								3			3
150	3										3
212	3										3
313			1	4	1	1			1		8
315	3	2								1	6
316	1										1
380.5	2										2
410							1	1			2
510									2		2
560									1		1
650							3				3
710									1		1
Total	12	2	1	4	1	1	4	4	5	1	35
%	34	5.6	2.8	11	2.8	2.8	11	11	14	2.8	100%

Table 43. 35 blind women, 16—41 yrs. of age, Undaan 1/1—1/7'65. Visual acuity less than 4/60.

	12	18	20	28	89	91	99	Total
110			1			4		5
122						1		1
150	1							1
190					1			1
212	1							1
313	1		3	1		2	3	10
315	3	1					4	8
316	5							5
380							1	1
410						1		1
530						1		1
Total	11	1	4	1	1	9	8	35
%	31	2.8	11	2.8	2.8	25	23	100%

Table 44. 58 blind men, over 40 yrs. of age, Undaan 1/1—1/7'65. Visual acuity less than 4/60.

	12	19	20	28	62	89	91	98	99	Total
110							11			11
150									1	1
212	2									2
313	1		4	1	2			2	3	11
315	3								4	7
316	1									1
410							13		2	15
420							1			1
530		1						1		2
640		1								1
650						3				3
710		3								3
Total	7	5	4	1	2	3	25	1	10	58
%	12	9	7	1.8	3.6	4.8	42	1.8	18	100%

Table 44 89 blind women, over 40 yrs. of age, Undaan, 1/1/1—1/7'65. Visual acuity less than 4/60.

	12	18	19	20	91	98	99	Total
110					34			34
212	2							2
313		1		16		1	3	21
315	2			2			3	7
316	1							1
370.1				1				1
380				1			1	2
380.5							2	2
410				3	12			15
530						2		2
710			2					2
Total	5	1	2	23	46	3	9	89
%	5.6	1.1	2.2	26	52	3.4	10	100%

Selection of patients for keratoplasty.

Patients who possibly could be helped by keratoplasty are included in the classification under type/site for conjunctival (2) affections and corneal (3) ones. The conjunctival site is to be taken into acccount also because the affection must have spread to the cornea, otherwise the patient would not be blind.

As I considered the patients individually, it became soon clear, that again certain groups would have to be discarded as candidates for keratoplasty, for the following reasons :

1. Too much corneal vascularization.
2. No healthy cornea left.
3. No visual acuity left.

Table 46. **Contra-indications against keratoplasty.**

MEN	0—17 yrs	16—41 yrs	Over 40 yrs.
1. Vascularized cornea	1	3	5
2. No healthy cornearest	4	3	3
3. Visual acuity: 0	2	5	2
Total	7	11	10
WOMEN			
1. Vascularized cornea	—	11	10
2. No healthy cornearest	2	3	4
3. Visual acuity: 0	5	3	0
Total	7	17	14

If these figures are subtracted from the totals of cases caused by conjunctival (2..) and corneal (3..) affections, we get:

Table 46a

	MEN			WOMEN		
	0—17 yrs.	16—41 yrs.	Over 40 yrs	0—17 yrs.	16—41 yrs.	Over 40 yrs.
Total conj. & corneal	18	20	20	12	26	35
Contra-indications	7	11	10	7	17	14
						minus
	11	9	10	5	9	21

In a total of 346 persons who had visual acuity less than 4/60 in both eyes, from this computation it appears that in only 30 males and 35 females keratoplasty could possibly be considered. As only the most obvious contra-indications have been applied, it is to be expected that on individual consideration of the cases the field will further be narrowed. In this group there are included 3 males and 6 females who are over 60 yrs. of age. In view of the fact that keratoplasty is still a complicated procedure especially if donor eyes are hard to come by, it stands to reason that preference should be given to younger people, who are still in the productive age group, and that old age as such can become a contra-indication. The figures given are ''maximal'' figures which as soon as it comes to practical application will certainly diminish.

Table 47 **Causes of blindness in keratoplasty candidates 1965. Figures in (parenthese) have less than 1/60 visual acuity.**

	Male			Female		
Cause	0—17 yr	16—41 yr	over 40 yr	0—17 yr	16—41 yr	Over 40 yr
Gonorrhoea	—	3 (2)	1 (0)	—	1 (0)	1 (0)
Keratomalacia	8 (7)	—	—	5 (4)	—	—
Syphilis	—	1 (0)	—	—	—	—
Trachoma	—	4 (3)	6 (4)	—	2 (1)	14 (6)
Tuberculosis	1 (0)	—	—	—	—	—
Variola	1 (0)	1 (0)	—	1 (0)	—	—
Trach/Variola	—	—	—	—	—	1 (0)
Miscellaneous	1 (0)	3 (1)	4 (1)	2 (1)	7 (1)	5 (4)

Comparison of the group studied 1958-1962 and that of 1965.

In the consideration of the usefulness of Keratoplasty only those patients blinded by conjunctival and corneal affections need to be taken into account. In the next Table the percentages for these categories are compared for the 1958-1962 group and for the 1965 sample. All conjunctival lesions are taken together. The other categories are : 311 and 313 taken together (simple corneal scars); 315 (leukoma adhaerens); 316 (fistula corneae); all other corneal affections taken together. Percentages are calculated with the totals from Tables B 1, 2, 3, 4, 5, 6. as hundred. For the group 1958-1962, children, the corrected figures are used, i.e. after elimination of those who on home visits were found to have died, or to have recovered sufficient eyesight to go to a normal school.

Table 48 Comparison of the percentages of conjunctival (2..) and corneal (3).. lesions in the group studied in 1958—1962 and that in 1965.

	Male		Female	
	1958/62	1965	1958/62	1965
0—17 yrs.	total 328.	total 23	total 248	total 22
2..	4.5%	—	5.6%	—
311/313	3.9%	8.7%	4.4%	13%
315	15.5%	17.5%	15.7%	9%
316	0.8%	—	0.5%	—
rest 3.. W	46.9%	52%	42%	32%
16—41 yrs.	total 1139	total 35	total 1091	total 35
2..	9.6%	8.5%	10.2%	2.8%
311/313	14 %	22.8%	17%	28%
315	28 %	17%	22%	23%
rest 3..	4.6%	5.7%	6.8%	2.8%
316	3.7%	2.8%	4.3%	14.2%
Over 40 yrs	total 942	total 58	total 991	total 89
2..	1.5%	3.6%	3.2%	2.2%
311/313	24 %	22%	26.7%	26%
315	16 %	13%	9.1%	8%
316	0.8%	2%	0.7%	1.1%
rest 3..	4.6%	—	4.6%	5.6%

From these figures it can be concluded, that the composition of the two groups, studied, that during 1958-1962 and the sample 1965, are sufficiently similar as far as the percentages of conjunctival and corneal causes of blindness are concerned, to allow us to use the calculations made for the 1965 sample for the 1958-62 group also.

For the 1965 sample we saw, that the following numbers of persons blind by conjunctival or corneal lesions could possibly obtain benefit from a keratoplasty procedure :

1965. from 18 boys under 17 yrs, 11 ; from 12 girls : 5.
from 20 men 17-41 yrs. 9 ; from 26 women : 9.
from 20 men over 40 yrs. 10 ; from 35 women : 21.

These are rough maximal figures. If applied to the 1958-1962 group, they would mean that ca. 35 % of all boys could profit by keratoplasty (50% of 70%). For girls this would be 34%, for blind men under 40 yrs. 30% ; for blind women under 40 yrs. 27% ; for men over 40 yrs : 30%; for women over 40 yrs : 22%.

These figures included : Trachoma, Leukoma adhaerens, fistula corneae and staphyloma corneae, which usually are considered unsuitable for keratoplasty.

As keratoplasty has two main drawbacks, viz. the scarcity of donor material, and the risk of downright "Bad" results (ca. 50%), it would be wise to select only the very favorable cases and those where visual acuity is very low, because in that way the chance for real improvement will be enhanced.

Selection of **favorable candidates** for keratoplasty.

If we approach the problem from the angle of selection of really favorable candidates, leaving out the "bad risks", which is not only justifiable, but even necessary in the present situation in this country, where donor material poses many difficulties, and where the general public still has to be won over to accept the new procedure, we will have to limit the application of keratoplasty for the time being to cases with uncomplicated corneal scars, i.e. the cases classified under the type/site numbers of 311, 312 and 313. The conjunctival numbers and also those of other corneal lesions are mostly connected with very serious corneal destruction where the results of surgery must be expected to be much less favorable than in cases with simple corneal scars. Trachoma, even if healed, will probably cause vascularization of the graft, so that the result of surgery tends to be disappointing.

If we exclude those with Trachoma as the cause of corneal lesions, and only consider simple corneal scars, we can calculate the following percentages for keratoplasty candidates for the groups under investigation :

Table 49. **Simple corneal scars, excluding Trachoma as a cause.**

age group	0—17 yrs	16—41 yrs	over 40 yrs.
1958-62 visual acuity less than $1^1/_2/60$.			
male	1.5%	5.7%	7 %
female	3.2%	5 %	6.4%
1965, visual acuity less than 5/60.			
male	8.7%	11.4%	14 %
female	13.6%	20 %	5.6%
1/8'66 — 1/8'67. visual acuity less than $1^1/_2/60$.			
male	1.7%	4.7%	7.2%
female	1.6%	7.2%	2.4%
1/8'66 — 1/8'57, visual acuity less than 1/10.			
male	1.3%	13.6%	5.8%
female	6.5%	18.9%	4.6%

If the surgeon who is to perform the keratoplasty procedure is willing to take the additional risk involved in operating on Leukoma adhaerens and Fistula corneae, we can calculate a further group of "not so good risks" :

Table 50. **Leukoma corneae and fistula corneae, excluding Trachoma as a cause.**

1958 — 62, visual acuity less than $1^1/_2/60$.			
male	16.9%	21.5%	7.9%
female	11.4%	20 %	7.2%
1965, visual acuity less than 5/60.			
male	17.3%	20 %	14 %
female	9 %	37 %	6.7%
1/8'66 — 1/8'67. visual acuity less than $1^1/_2/60$.			
male	16.9%	24.5%	7.9%
female	11.4%	20 %	7.2%
1/8'66 — 1/8'67, visual acuity less than 1/10.			
male	17.7%	16.3%	6.2%
female	10.3%	12.3%	4.2%

Comment. Whether the limit of visual acuity is put at $1^1/_2/60$ or 5/60, the percentage of candidates for keratoplasty, as well the "good risks" as the "not so good" risk is less than 25% of corneal and conjunctival lesions for children and less than 30% for adults. However, even if these figures certainly do not give reason for high hopes for an early significant diminution of blindness, and even if we do take into account the fact that these percentages on individual scrutiny will prove too high, the fact remains that a certain number of blind persons CAN be helped by keratoplasty, making it necessary to put things in motion for its application.

Difficulties. The first and greatest difficulty is getting donor material. On various occasions where by publicity in the press on the possibilities of the procedure (sometimes too optimistic) a number of patients came, asking for surgery, but there was until now nobody who OFFERED his eyes to be used after his death, not even on a tentative basis. The general public is not yet prepared to take this step, so the supply of donor material is very limited. Thus the utmost care in the selection of patients for surgery is necessary so as not to waste material, and not to disappoint the patients who are operated. For these reasons the situation has to be judged realistically, and a slow start has to be made with a very carefully outlined policy, in which full advantage is taken of technical progress in the field, adjusted to the possibilities as they exist in this country. It is the present policy (455) to limit candidates for surgery to persons with visual acuity of no more than 1/60, light projection intact, and to concentrate the actual surgery in the larger centres.

It is hoped that slowly, as successfully operated cases get into circulation, the number of people who offer their corneas for use after death will increase until the supply is sufficient. This is one of the reasons that make it important to operate on the "good risks" first, so as to give the method a good name among the public. Religious objections to the use of material from dead bodies can be countered by expert opinion from all the major religions (Heath 170)

Conclusions. 1. According to our above figures keratoplasty could possibly be useful for at most 25% of blind children, and 30% of blind adults.

2. On closer examination of patients individually these percentages will prove too high, but they are still worthwhile.
3. In view of the very limited supply of donor eyes, the indications for keratoplasty should be sharply limited.
4. Preference should be given to patients which are considered "good risks".
5. The present limit of visual of no more than 1/60, light projection intact, is realistic.
6. Future press publicity in this field should stress the need for more donor material by voluntary donation, less on the hopes for helping "all blind people", because disappointment of many blind people is then unavoidable. The burden of the blind persons' life is heavy enough as it is, it should not be added to by misleading information, raising hopes which cannot be fulfilled.

Chapter Twenty Eight

PREVENTION OF BLINDNESS.

There are three factors involved in preventing blindness:

1. The ophthalmologist who understands the problem,
2. The general public which should learn to understand enough about health problems to follow simple precepts to preserve health.
3. The public health authorities which should take appropriate measures to safeguard the health of the people.

Thus, according to da Silva (444) the problem was concisely put by Park Lewis. In the previous chapters an effort has been made to analyze the causes of blindness in and around Surabaja (East Java) with the following results in the various age groups :

A. For children the most frequent cause is Xerophthalmia in its severe form. It also gives its special Indonesian signature to the morbid picture of general malnutrition, to which it is often added. Poorwo Soedarmo (379) gives as the cause of malnutrition as seen in Indonesian infants :

1. Decrease in breast milk production after 6 months, with ensuing growth retardation.
   Inadequately regulated additional feeding, enhanced by superstitions, precluding a rational baby diet.
3. Incorrect weaning pattern.
4. Lack of understanding for the close relations between food and health. If an infant fails to grow, the cause is attributed to "teething", "growing pains", "worms" etc. but not to insufficiently correct feeding.
5. In young children increasing exposure to environmental factors e.g. infectious diseases enhances their chances to incur damage to the nutritional status.
6. The correct care of small children is often known too little or not at all to the mothers, who rely on custom and oldfashioned habits, i.e. the local "adat".

All of these factors together prevent young children from receiving a diet with sufficient essential nutrients, especially Protein, fat and vitamins (among them vitamin A) causing the pattern of general malnutrition with Xerophthalmia usually encountered in the Indonesian context.

To prevent this unfavorable situation it is firstly necessary to see if enough foodstuffs are available to feed all children adequately. Animal protein, especially milk and eggs being scarce and expensive, which cannot be altered in a short time it is necessary to use a balanced mixture of vegetable proteins instead. As in many regions green foodstuffs are available in abundance it should be possible to compose a satisfactory locally adapted diet in each region and recommend its use. Red palm oil would be of great help as it contains fat and a high carotene-level at the same time. If the fish oils could be made available they would contribute fat and vitamin A of a high quality. Whether there would be enough for everybody is a question of great importance which I cannot answer, but that the available resources are not used to their full capacity is certain.

The second most important cause of blindness in children are hereditary and congenital affections until now defying prevention. From advanced countries (Denmark, 452) there are reports on efforts to prevent the continuance of hereditary traits by premarital counselling but for Indonesia the problem is not yet in order.

The third most important cause of blindness in children is conjunctivitis gonorrhoica, more often in older children than in the newborn. It has been proven that in countries, where the method of Credé (nitras argenti eyedrops as soon as possible after birth) has been applied rigorously conjunctivitis neonatorum can be prevented 100%. Unfortunately the larger part of conjunctivitis in older children can only be prevented by higher general hygienic habits, and preferably by eradication of gonorrhoea generally, which is a notoriously difficult undertaking which until now has not met with success in any country. By an effective organization of MCH centres it would be possible to prevent malnutritional blindness and conjunctivitis neonatorum at the same time, as well as congenital syphilitic blindness.

B. Productive age: 16-40 years. Different from the first age group in the age group 16-40 years, the most frequent blinding diseases are: Trachoma, Syphilis and conjunctivitis gonorrhoica. The eradication of Trachoma is a very difficult problem, which seems to be in the process of solving itself! The only rational method of prevention would be mass treatment of the whole population in an area, not only of schoolchildren. The experience from many modern reports points to the importance of repeated reinfections for the development of severe Trachoma, which makes the treatment of schoolchildren only who themselves do not yet suffer from severe Trachoma, selfdefeating as long as sources of reinfection are not treated at the same time. It seems that by a slow rise in general hygienic ideas and by treatment of the severe cases, the opportunities for reinfection are already diminishing without any special effort on a large scale. All kinds of medical personnel can be trained to treat Trachoma now (sulfa orally and antibiotics locally will soon convert a severe Trachoma into a non-infectious stage) which at the time seems the cheapest and most effective measure which can be taken. The cost of a large scale vaccination campaign would be prohibitive, and until now of doubtful value.

The prevention of Syphilis and Gonorrhoea does not lie in the province of the ophthalmologist, except in an effort to prevent serious damage by tabetic optic atrophy. At one time Sobanski (453) has developed the theory, that the atrophy of the optic nerve depended on the imbalance between a relatively high intraocular pressure and the supposedly low general blood pressure in tabetics. He even performed antiglaucomatous surgery in tabetics to prevent this development. Other authors (i.a. Constantine, 61) have not generally confirmed his theory, which is now completely obsolete. Klauder et al. (207) have recommended a close cooperation of dermatologist and ophthalmologist, and regular careful check-up of visual field and fundus in known syphilitics, in an effort to detect early signs of optic atrophy indicating renewed treatment. This would be very useful, but it is in the situation as it exists here and now still rather difficult to organize, with a scarcity of doctors and the general carelessness of patients.

C. Oldest age group, over 40 years. In this age group the blinding diseases of the productive age are still active, and two new frequent causes of blindness are added, viz. Glaucoma and senile Cataract. The latter can only be treated, not prevented, until now, and somewhat the same goes for Glaucoma, as they are essentially diseases of aging. The difference is, that whereas Cataract can better be treated after it has caused a definite visual impairment, Glaucoma MUST be treated early to be successful. This difference and the danger involved in postponing treatment of Glaucoma is not sufficiently known, and it would be necessary that all kinds of medical personnel, and also other authorities, like village heads, teachers etc. would know about it, and advise older people with visual complaints, to see an oculist as soon as possible. It would be helpful if everybody needing reading glasses would be seen by a doctor first, and still better if the patients would not change their doctor for every new complaint. This prevents any one doctor from regularly measuring the intraocular tension and detecting early signs of a rise in tension. As it is now, cases of Glaucoma and optic atrophy do come to the oculist far too late, because the symptoms are ascribed to cataract. Much could be gained if this misunderstanding could be corrected.

Public Health authorities will be primarily interested (and rightly so) in causes of blindness in children. One of the measures which can be taken in the fight against important diseases is to make them mandatory reportable. This has been done in the case of Xerophthalmia in Jordan (WHO Report, 570) to facilitate the study of the disease. In some states of the USA blindness as such must be reported to the public health authorities, which enables them to make an accurate assessment of the relative importance of causes of blindness, and to help those who need help, either medical, educational or financial. Public Health authorities could also enforce measures like e.g. refusing admission to school for children with Trachoma, but this special measure is not to be recommended, as usually the source of infection with Trachoma is the family, not the school, and with a few days' treatment children can now be brought into a non-infectious condition. Enforced reporting and treatment of venereal disease has variously been applied, but not always with success (Simons, 448). The way of persuasion, education, propaganda, improvement of water supply, of garbage disposal etc. is by far to be preferred to that of enforced reporting and treatment in the fight against blinding diseases. In industrialized countries the stress will partly be laid on prevention of accidents during work (Vaquez Barriere, 530) and on careful treatment of diseases like diabetes and vascular disease, in an effort to postpone ensuing blindness (Velhagen, 535).

Prevention of blindness in Indonesia.

Indonesia as a large and varied archipelago has special difficulties in preventive medicine. Firstly : a collection of many larger and smaller islands is always a notoriously difficult problem in public health. It is nearly impossible to disperse enough doctors and other medical personnel to bring each inhabited islet within easy reach of medical help. Secondly : the large size and variety of the country make it necessary to decentralize public health measures to a certain degree. What is right on one island, can be wrong in another one. Blindness and its causes should be studied in certain circumscript regions, and measures of prevention should be devised for that region, taking into account local possibilities, local custom and the local economy. Generalized measures for the whole country would be less practical. In postwar years there have been several conferences devoted to the study of the problem, and although the sequence varies, the diseases which were considered the most important were always the same : Konperensi Dinas Pembarantasan Penjakit Mata, 1958, (211) puts them as : conjunctivitis, Trachoma, Xerophthalmia, in this order, whereas the ophthalmological section of IDI, 1958, (186) ranks them : Trachoma, Blennorrhoea, Keratomalacia. At the Seminar at Bandung, 1964 (518) on Blindness, the order was : A-deficiency, Trachoma, Gonoblennorrhoea. All three of them are still important in East Java, but I would like to add : Syphilis and Glaucoma. In devising preventive measures against them, it is selfevident, that cooperation with many other medical branches is necessary, especially in the fight against Xerophthalmia and Venereal Disease. Trachoma and Glaucoma should be a job for the general physician and the oculist between them, always helped and supervised by public health authorities The education and training of all types of medical and paramedical personnel should always take into account the importance of preventing blindness, and it would be a good thing, if e.g. the most important preventive aspects of ophthalmology were incorporated into the curriculum of nurses' and midwives' training and taught by ophthalmologists, not just in a desultory way by teachers in other disciplines.

The question : what is the cost which should always be put and answered, can in this case be answered as : not so very much, compared to what can be gained! Most of the measures which would be needed for making the fight against blindness more effective, can be built into existing public health schemes. The fight against malnutrition could easily give enough attention to the aspect of vitamin A-deficiency, to make it effective against the scourge of Keratomalacia. The fight against venereal disease could possibly draw new inspiration from the urgent demand from the oculist to do something against it. The fight against Trachoma is a fight for better hygienic standards in a very general way. Expensive the prevention of blindness need not be, it only would need a more general awareness of the fact, that most public health measures by a slight bit of special attention could be made effective for the fight against blindness in addition to their general purpose. Instead of new expenditure it would be possible to save high sums of money which are now wasted unobserved by loss of health, loss of working ability and high costs of social services to the blind and weak-sighted.

Chapter Twenty nine.

The Role of the Oculist in the Rahabilitation of the Blind.

It is the sad experience of every oculist, that a certain number of patients cannot be helped any more in a medical sense. As new medical and surgical methods slowly are developed, there is a number of types of blindness coming into the sphere of curable blindness, but at present there still remains a hard core of kinds of blindness which cannot be cured, and from the figures in previous chapters it can seen, that their number is still high, and from the chapter on Keratoplasty can be concluded, that only a small percentage of them eventually can possibly be helped. The fate of these blind people depends on the help they can get from the seeing. According to Schubert (420) in socalled primitive societies the struggle for life is so hard, that probably the blind do not get enough help, and consequently die early. In more advanced societies the blind are helped, but often not yet optimally. But probably not only the amount of help they receive from the seeing, but also factors which could be either psychologically or physiologically related to blindness play a role, as it was found by Singer (1929, 449) that mortality in the blind is 2-3 times higher than in the seeing population (comparable to tuberculosis, at that time) although the blind do not as often die from accidents as the seeing. A number of the blind is too old to be re-educated for any type of work, or even for any kind of recreation. They have to be helped in their daily life and the most which can be done is to teach their families to help them as efficiently as possible.

For the younger age groups on the other hand, it is possible to be trained for work which comes within their scope, and to be taught certain skills which can provide recreation and relaxation, like music and reading.

In a country where the general public not yet realizes how many blind people there are, and still less what can be done to make their life more useful and agreeable, the oculist is the keyfigure in the field of rehabilitation. He knows at first hand the extent of the problem and should inform himself on the possiblities for rehabilitation which are available or which should be made available. He should then try to induce others to undertake the various jobs which are inherent in rehabilitation of the blind, serving as a link, if need be as a motor, to estabish contact and progress in the field.

There is a slogan : a blind person is exactly like every other person, only he is blind. This is slightly misleading: physiologically and psychologically a blind person is not exactly the same as a seeing person. The fact that a totally blind person never undergoes the stimulant of daylight is sufficiently important to alter his various rhythmic physiological functions, which e.g. is evident from the difference in eosinophilic cell count, which in a seeing person shows a diurnal rhythm, which lacks in blind persons. Many other rhythmic diurnal functions, like e.g. waking and sleeping are not the same in blind and seeing people. Especially in a person who is blind from birth, it is evident, that his experience, which is after all the material from which the psyche must collect much of its inducement for development, is quite different from that of a seeing person. It is not doing the blind any good if they are taught that they are exactly the same as seeing people: like seeing people also, they must learn to live with their limitations, which in their case are rather serious. Unfortunately they cannot even have the same opportunies as seeing people, precisely because of their handicap. They can have the same rights, but they must realize that the rights of seeing people are not always honored too, and that in the implementation of basic rights for everybody there is still much to be hoped for, little which is already fulfilled.

On the other hand the general public should learn to realize that also blind people can be valuable members of the community, that their handicap is serious, but can largely be overcome with expert help, so that many blind persons are able to earn their own living and contribute to the life of their community. To a certain extent they will have to be dependent on the help of the seeing.

For the problems involved in the rehabilitation of the blind it is necessary to divide them in age groups, and to take into account the age at onset of blindness.

Children. Blind children whose intellect is normal, can be educated in special schools for the blind, where the normal curriculum of other schools is followed, adapted to the abilities of the blind by using Braille in reading, writing, arithmetics, and where certain other aids, e.g. relief maps are used as far as necessary. Another possibility is the so-called integrated system, which places blind children in normal schools, but allows them to use special aids, Braille etc. and where they are helped by specially trained teachers for the blind in subjects which they cannot pick up easily in the normal classes. Both of these systems are however dependent on a certain pre-school training of the blind child: they must be able to help themselves in the matter of eating, dressing and toilet. Most parents of blind children do not know how to train blind children in these simple but essential skills, and in this field the oculist can be of much help, in that he establishes as early as possible the necessary contact with either a specially trained social worker, or another type of counsellor, who can advise parents on these points. Young blind children often appear retarded, they may develop odd habits, so-called blindisms, e.g. the digito-ocular reflex (Franceschetti 126) and they tend to be inactive, as their curiosity is not stimulated as it is in a child with normal eyesight. The parents have to learn to train their blind child in using its other senses with special games, singing, story telling etc. to make up for the lack of visual experience. Very intelligent parents can maybe find their own way in this difficult field, but most will need guidance. Many of them have first to learn to accept their child's handicap, and to overcome the inclination to search for the guilty person. Here again the oculist can be of help, by explaining the cause of the child's blindness, making it clear that this is not the "fault" of either of the parents, or of some outside "evil influence" as is often presumed: this idea gives rise to much resentment, or to a feeling of shame, both of which are unfounded and misplaced, and do only harm to the blind child itself. It is for a blind child at first not at all clear that it is different from the others, but as it slowly realizes this, it must be tried to get it to accept the situation. This can be brought about by parents who themselves have accepted it, but not as long as they remain resentful. Rehabilitation can only begin, as well in the case of the parents as in that of the child, as soon as the handicap is accepted, and the decision is made to make the best of it.

That the best can be very good is already shown by the careers of many blind people, who have attained positions in society which are useful and honored: mostly they have been helped by a devoted parent, or other helper, but their own gifts have made success possible.

As soon as an oculist discovers that a child will be blind for life, he should put things in motion to begin the long road of the child's education. If there is a school for the blind in the neighborhood, this could be contacted, and asked for counselling of the parents, or at the Eye department itself a counselling service could be set up. The important point is that the parents need not have the feeling that they are left to themselves in their difficulties, and that the "retardation" of the child's development is prevented as far as possible. From the degree and type of blindness the oculist is able to provide the counsellor with data on what the child can be expected to do: children blind from birth are different from children who have had some visual experience, totally blind children differ from those with a visual rest. Blind children should be examined also for additional handicaps: deafness in one or both ears or neurological defects, and treated and handled accordingly.

All of these data as far as relevant should be given to the counsellor, and later to the educator so as to make use of the remaining faculties as efficiently as possible, and so as not to overburden the child setting him tasks which he cannot possibly fulfil.

It would be useful to give the educators of blind children some extra training giving them an idea of the different types of blindness and near-blindness, so that they neither do damage by over-nor by understimating the capabilities of their charges. It is senseless to blindfold a child with some visual rest in order to force him to use his fingers instead of his eyes, and on the other hand e.g. highly myopic children should not be allowed to read for long periods at a time but with frequent intervals for other work inducing them to look in the distance. The oculist who sends a child to a school for the blind should realize what teachers are doing, and give them advice on what types of training are useful and not harmful for each individual child. On the other hand schools for the blind should not accept blind pupils before they have been examined by a competent oculist: if this is not adhered to there is a chance that children are considered blind, and educated on that basis, who really can be helped with either an operation or glasses, so that they can go to a normal school. Even in some western countries this principle is not always adhered to.

U. Beermann (1964, 233) explains the mistakes which were made in Germany at the time before there was a distinction made between really blind and weak-sighted children. After a cooperative effort by oculists and pedagogues the various types are differentiated first, and on that basis is tried to find the most useful kind of schooling. An important angle which has to be considered is the prognosis of children with a visual rest: if it is to be expected that vision will deteriorate still further, it can be advisable to start the child on Brialle soon, as learning is much easier at a young age, but if the prognosis is dubious or even good, the teaching of Braille can be psychologically harmful because of its implications of permanent blindness. The oculist should consider all of these facets and shape his advise accordingly.

Young adults. It is evident that in the case of young adults who have recently become blind all of the foregoing factors should be taken into account also. For them it depends on their past history, what will be the best type of rehabilitation. If they have been to school, and cannot continue, they have to be trained in "being blind". They must relearn to help themselves in all daily necessities, which can be facilitated by providing them with special aids, like Braille watches, special eating plates to prevent slopping, special drinking cups, and in the case of housewives needles with a surgical eye etc. Many old skills can be relearned with slight adaptations, but expert help is essential to attain the best result, and if this is not available the oculist should do his best to stimulate the inception of appropriate services for the blind.

Older adults. Much of what is said for young adults is valid for older adults too, as long as they are not too old to learn at all. Members of the family of all blind people should be taught the simple tricks of helping a blind person unobtrusively. This includes e.g. walking arm in-arm with him, not pushing him from behind and thus letting him take all the risks ; warning for steps ; calling attention to special landmarks; so as to make it possible eventually that the blind can walk certain routes alone ; not moving the furniture around without warning him ; not leaving doors ajar, but either shutting them or leaving them wide open, to prevent collisions of the blind person with a half open door ; guiding him to a seat by guiding his hand to it ; addressing him by name if more people are present ; saying something on entering a room where a blind person is present so as not to surprise him unexpectedly ; all of these "tricks" are quite simple for everybody to learn, but they can make the difference for a blind person between always feeling slightly unsafe and feeling at ease. The family should also be told, that many blind people can be valuable workers, and that they probably are much happier if they can work, especially if they feel that their work is good and is appreciated. Many people feel so much "pity" for the blind that they will not allow them to do anything at all, which is the first obstacle in the rehabilitation of the blind.

If the blind person can be provided with a white walking stick, he can be trained to go out alone, using the stick as well for his own orientation, "feeling his way", as well as to give warning to the seeing. It would be useful if the white stick would be officially recognized as the attribute of the blind (possibly also of the deaf) and if in the training and examination for the driver's license attention would be called to it, as well as e.g. in the press and in schools.

As soon as a recently blinded person has learned "to be blind", he can be taught Braille, especially if he has been to school formerly and so can spell, and depending on his abilities and inclination he can be taught skills that will either enable him to continue his old job, or to learn a new one if necessary; here again the oculist should advise the vocational teachers and officials involved in job finding for the blind on the visual abilities of individual cases, so as to make as much use of visual rests as possible and anyway not to do damage to them. Close cooperation with the Social Services who are working in the field would be very advisable.

In the case of young and older adults the oculist should realize two points : first, blindness at first is often the cause of depression, although this rarely seems to lead to suicide, if there is no other psychiatric condition present (G.R. v.d. Berg: NTvG **111:** 312, '67) Only after the depression has cleared up the patient can listen to proposals for rehabilitation, there is no sense in trying to hurry things along in this direction.

The second point is, that often in cases of blindness after eye disease the patient and his family will unconsciously consider the doctor at fault for not having been succesful in treating him. This makes it maybe advisable for the oculist not to start talking on rehabilitation himself, but to let other workers in the field start it. The task of the doctor in these cases is to judge how far the mood of the patient is receptive for the idea of optimal rehabilitation and to see that a start is made. To be able to do this, he must be able to rely on competent workers in the field of rehabilitation, and if these are not yet available he should do his utmost to seek interest for the problem from authorities and the general public : rehabilitation of the blind is just as important as preventing blindness, as well in a generally social as in an economic sense, and the oculist is the one who knows the extent and the possibilities for solution of the problem, and so often will have to be the prime mover, also if he does not do the true work of rehabilitation himself. He should always be interested in it and involved in it, as the root of the problem is a medical one where the laymen who are competent in the social and technical work need essential advice from the oculist.

Chapter Thirty.

SUMMARY AND CONCLUSIONS.

Over a period from 1/1'58 till 1/1'63 and another period from 1/8'66 till 1/8'67 all patients seen at Undaan Eye Hospital who were finally blind were classified as to causes of blindness, using the International Standard Classification for Causes of Blindness.

The rate of blindness for East Java was estimated to be ca: 250: 100,000 inhabitants.

The type of investigation was based on notes made on all blind persons of all ages who came to the Outpatients' Departement of Undaan Eye Hospital at Surabaja.

Definition of blindness used: visual acuity of 1/60 or less in the better eye after correction of refractional errors.

Methods and difficulties in history taking, and in diagnosis of causes of blindness are explained and methods of examination described.

The frequency of blind patients among the total outpatients showed a small decrease between 1958 and 1962, which did not hold up in 1966/67. There was found a high frequency of blindness in very young children, a low frequency of new cases of blindness between ages 5 and 20, a slow rise after that age. Tables and curves are given for the frequency of blindness with age and compared to the composition of the age groups in the general population.

The degree of blindness was determined for the year 1960 only, and compared to data from England. The degree of blindness in East Java was distinctly higher than in England (more people were totally blind without any visual rest).

In chapter 8 the International Standard Classification for Causes of Blindness is given with a Manual on its use.

In chapter 9 the adaptations which I made in using the Classification are explained.

Chapter 10 gives the cross-tabulations for each year of the study, divided into 3 age groups (0-16 yr, 16-40 yr and over 40 yr) by sex.

For the period 1/8'66-1/8'67 not only patients blind by using the limit of visual acuity of 1/60 are given, but also persons who were blind if the higher limit of 1/10 for visual acuity were used.

From the cross-tabulations percentages were calculated, arranged in the same type of tables as those given by Sorsby in his latest publication on causes of blindness in England, and comparisons are made between causes of blindness in Indonesia, East Java, and England, in their relative importance.

Distinct differences emerged: in East Java in children Xerophtalmia and Conjunctivitis gonorrhoica were important causes of blindness which did not appear at all in the lists from England. In the older age groups Trachoma, Conjunctivitis gonorrhoica and Syphilis had a higher frequency in East Java than in England. In the oldest age group in England vascular disease and Diabetes are frequent causes of blindness, which as are yet negligible in East Java. An analysis of the reason of various differences is tried.

Chapters 15 and 16 are devoted to results of former investigations in Indonesia and in neighboring countries.

In chapters 16 throught 23 several causes of blindness are analysed, especially the existing differences between these diseases as they are seen in tropical countries and in pigmented races, and as they are discribed in the textbooks of ophthalmology.

Conjunctivitis gonorrhoica is still an important cause of blindness in all age groups, as well as Syphilis. Their prevention depends on the fight against venereal diseases in general, but in the case of conjunctivitis gonorrhoica much could be gained by more cleanliness and by keeping articles used in washing and sleeping separate for each member of the family.

Trachoma, although still an important cause of blindness, is not as frequent as it has been, and especially the severe cases become rarer, without any special effort in public health measures, except treatment.

Glaucoma appears to be more frequent than has been thought in former times, even showing a percentage of 30% in the oldest age group in women. It would be useful if all types of medical personnel were to be educated in the existence and dangers of this disease, in order to create the opportunity for starting treatment early enough to prevent dangerous sequelae.

As Keratomalacia, the severest stage of Xorophthalmia, was shown to be the most frequent cause of blindness in children, 2 chapters were devoted to it : in chapter 24 an introduction based on findings of others is given, with special attention for points which lie within the field of ophthalmology, but which have not yet been completaly explained (foam in Bitot's spots, nightblindness, long eyelashes, influence of strong light). In chapter 25 a special study is reported which was made during two cousecutive years on all cases of Xerophthalmia seen at Undaan, with an analysis of incidence with age, sex, season, previous diseases.

Examinations of a pediatrician were summarized in using the malnutrition index of Oomen for several hundred cases. By home visits various socio-econimic factors were ascertained. Proposals for prevention were reviewed.

As there had arisen a suspicion that the mortality of small blind children possibly was high, in chapter 26 a special study into the fate of small blind children is reported. Inside 2-3 years 30% or more of these children had died and a large number had been brought to a village. This makes an efficient education for these children very difficult.

An effort was made in chapter 27 to estimate how many blind persons could eventually be helped by Keratoplasty. The percentage was disappointingly low, but still worthwhile.

In chapter 28 various aspects of the prevention of blindness were reviewed. Chapter 29 gives an exposition of the role of the oculist in the rehabilitation of the blind, which is especially in a devoloping country a key-role. A list of references is added at the end of the study.

USE OF THE INTERNATIONAL STANDARD CLASSIFICATION IN THE PRESENT STUDY.

In the present study the International Standard Classification for causes of Blindness was applied as far as possible according to the manual belonging to it. The book of Mrs. Schappert-Kimmijser was based on the same system with minor differences (414) and Sorsby in his review on Incidence and Causes of Blindness 1948-1962 used more or less the same classification, but he changed some numbers around, so that it is necessary to compare his list of numbers carefully with the international list.

On some problems I had to decide which way would be most practical among which :

1. Blindness fron different causes in the two eyes.

If it was not clear which eye had been blinded last and thus should be the decisive one, I assumed that if one eye had the classification 99/150, which is the most ambiguous classification, this would be the first eye, and I used the classification of the other eye. If there was not much essential difference in interest between the classification according to site and type in the two eyes, I classified it under the site/type of the right eye, assuming that in this way the division would be as even as possible. On each table I placed an additional small group of cases, where classification seemed difficult and I gave a compilation of these cases on the basis of taking each eye separately (table B. 7). In that way a number of cases of some interest were not mixed up in the large lists, but kept separate.

2. Double etiology.

The manual of the International Classification prescribes one etiological factor to be classified only, but in a number of cases I prefered giving 2 etiological factors, e.g. where it was not at all certain which had been the decisive one.

3. To classify some factors which seemed important to me in this country.

I **added some special numbers** (as is allowed):

for classification by etiology, I added **20.1,** meaning Trachoma and Wassermann test positive.

28.3: Koch-weeks bacillus found in smear, and probably the cause of blindness.

28.4: Morax-Axenfeld diplobacillus found in smear, and probably the cause of blindness.

65.82: Hydrocephalus.

65.83: meningo-encephalitis (diagnosed by neurologist)

98: under this number I brought all cases where Wassermann test had been done, but was negative.

To the numbers for classification by site/type of affection I added :

190: was always used for excised eyeball.

315: Leucoma adhaerens.

316: fistula corneae.

370.1: localized keratectasia at site of pannus trachomatosus.

380.4: Pterygium.

380.5: Staphyloma corneae.

4. Unusual combinations.

Of etiology and site/type of affection. If 2 factors, one etiological and the other topographical, were present, which were blinding by their combination, I classified a case by them, even if the combination was unusual. E.g. normally cataract will be classified as 91/410, but if also Trachoma was present which prevented a successful operation I classified the case as 20/410. The unusual combination 20/110 was also used, with a view to the possibility that Trachoma is an etiological factor in the development of Glauçoma. 20/122 means, in this way, that a case of Myopia could not be corrected because of sequelae of Trachoma which were also present.

5. Value of diagnosis.

Of the most important diagnostic categories the trustworthiness can by judged by the following considerations :

12. Gonnorrhoea: diagnosis by conjunctival smear or explicit history.
19. Syphilis: Wassermann test positive and confirmation by experts from the Institute for Venereal Diseases, where all patients with positive serological tests were seen.
20. Trachoma : usual diagnostic signs.
21. Tuberculosis : confirmed by specialists from Tb Centre.
65. Specific neurological diagnosis was mostly made by specialists from the Faculty of Medicine of Airlangga University.

6. Hereditary diseases.

Because of the lack of compulsory registration of the population and because of the often complicated family connection related to Islamic marriage law, it was not practicable to investigate diseases which could be hereditary, except in a few obvious cases.

7. Tabulation.

For the purpose of giving as much relevant information as possible, the blind patients were divided by age and sex. Because of the relative uncertainty of the age of the patient as stated, the division in **age groups** was made according to the most important phases in life :

0-17 yrs; 16-41 yrs, active working age; over 40 yrs : older age. According to Dr. R.H. Su'dan (1967. 488) life expectancy at birth in Makassar is ca. 42 years at present. At 40 yrs. of age Presbyopia generally is to be corrected with glasses of 1 dptr. The general opinion among the population at large is, that people over 40 years are "old". These various factors led me to choose 40 years as a borderline.

For each year and each group **cross tabulations** were made, giving the classification by etiology and by site/type of affection (all Tables marked A). For the 5-year period 1958-1962 the data for each group were compiled into one cross tabulation (all Tables marked B).

Tables for patients seen in the year between 1 Aug. '66 and 1 Aug. '67 were made in the same comparable way, but using 2 different definitions of "blindness" one with limit of visual acuity: less than $1^1/_2/60$, as in the 1958-1962 period, one using as a limit : less than 1/10.

The data given in all of these Tables were then arranged and calculated in the same way Sorsby has tabulated his data in his 1966 report. (All tables marked C).

For easy comparison some of the relevant Tables from Sorsby's report have been inserted, if necessary after slight adaptation ; e.g. Sorsby's Table 9 which shows 7 age groups has been recalculated to reduce the data into 3 age groups, more or less parallel to those used in the present study.

Trends.

The purpose of adding data on patients seen in the period Aug. 1st —'66—Aug. 1st '67 was to see, if there was to be detected any difference in incidence/and or causes of blindness between the early period and the later one. The incidence of blindness, calculated from the number of patients seen in the Outpatients' Department showed only a slight decrease from 5.5% in 1958 to 4. 6% in 1962 and again 4.8% in 1966/7.

For the most important causes of blindness where a "trend" in incidence could be expected, i.e. for children : conjunctivitis gonorrhoica and keratomalacia, and for adults : conjunctivitis gonorrhoica, syphilis, Trachoma and Glaucoma, these were calculated, as will be shown in the relevant context (chapter fifteen).

Monocular blindness.

Data on patients, who were only blind in one eye, were collected during the period Aug. 1st '66 — Aug. 1st '67 only, again using 2 definitions of "blindness", one with visual acuity less than $1^1/_2/60$ and the other with visual acuity less than 1/10. From an ophthalmological point of view these figures are of some interest. According to the manual of the International Standard Classification of Causes of Blindness these patients are NOT to be included, of course, in the large tables, but it seemed interesting to give them separately, just to give a complete picture of "blinding" diseases. As there was not much difference in the division in age groups between monocular and binocular blind patients, the ages of the former were not given in detail.

The calculation for monocular blind patients according to the C-type of tables was limited to the most informative ones.

Table C 1. Blindness from the same cause in both eyes, Undaan, 1958-62, visual acuity less than $1^1/_2/60$. (Compare Table 7, Sorsby, p. 10 and p. 11).
Classification by site.

	0—17 yrs		16—41 yrs		over 40 yrs		Total	
Eyeball	77	11.4%	321	14.6%	494	25.5%	892	18.7%
Conjunctiva	36	5.3%	222	10.1%	48	2.5%	306	6.2%
Cornea	425	61.5%	1139	52%	865	45.6%	2429	51.1%
Lens	49	7.2%	46	2.7%	186	9%	281	5.9%
Uveal tract	28	4.1%	213	9%	155	8.3%	396	8.3%
Retina	9	1.4%	38	1.7%	48	2.5%	85	1.7%
Optic nerve, optic pathway visual centres	45	6.7%	196	9%	101	5.3%	342	7.1%
Other	5	0.7%	11	0.5%	6	0.3%	22	0.4%
Total	674	100%	2186	100%	1893	100%	4753	100%
Classification by etiology								
Infectious *	79	11.7%	935	42.3%	756	40.4%	1770	37.2%
Trauma	1		14	0.6%	7	0.4%	22	0.4%
Systemic disease	316	46.9%	24	1.1%	17	0.9%	357	7.5%
Prenatal influence	121	18%	52	2.3%	21	1.2%	194	4%
Undetermined	157	23.2%	1161	53.1%	1092	58.3%	2410	50.7%
Total	674	100%	2186	100%	1893	100%	4753	100%

Table 7 from A. Sorsby: "Incidence and Causes of Blindness in England and Wales 1955-1962" 1966, p. 10 and p. 11.

58, 272 patients, 1955-1960, same cause of blindness in the two eyes, by site and etiology.

Site. p. 10	
Eyeball	15.5%
Conjunctiva	0.1%
Cornea	2.3%
Lens	23.9%
Uveal tract	11.4%
Retina	41.0%
Optic nerve	4.9%
Vitreous	0.1%
Multiple, ill defined	0.0%
Globe normal	0.8%

Aetiology. p. 11.	
Infectious diseases	0.5%
Trauma	0.4%
Poisoning	0.3%
Tumours	0.9%
Systemic diseases	12.7%
Prenatal influences	5.4%
Undetermined	79.8%

Chapter eleven.

CALCULATIONS FROM THE PREVIOUS TABLES.

Percentages for different kinds of causes have been calculated from the Tables marked B in the previous Chapter. From the additional cases in the A Tables, those caused by the same etiology, but different site, in the two eyes have been included in the calculations of the following C-Tables, whereas those from different cause AND different site are collected in a separate Table B 7.

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*) Note: Infectious diseases: excluding transmitted maternal infections.

Table C 2. Clinical entities responsible for blindness, Undaan, 1958-62. Visual acuity less than $1^{1}/_{2}/60$. Same cause in both eyes. All ages by sex. (Compare Table 8, Sorsby, 1966, p. 12).

Clinical entity.	Male		Female		Total	
Congenital defects :						
Globe as a whole	37		28		65	
Lens :						
Cataract	33		24		57	
Dislocation	3		1		4	
	36		25		61	
Uveal tract :						
Malformations	6		4		10	
Congen. syphilis	1		2		3	
	7		6		13	
Retina :						
Retinoblastoma	2		5		7	
Optic nerve:						
Optic atrophy	3		3		6	
	85	3.4%	67	2.8%	152	3.1%
Abiotrophic defects:						
Corneal dystrophy	2		3		5	
Retinit. pigmentosa	27		18		45	
Macular dystrophy	—		3		3	
	29	1.1%	24	1 %	53	1 %
Glaucoma	197	8 %	401	17.3%	598	12.3%
Interstit. keratitis	10	0.5%	12	0.5%	22	0.5%
Senile cataract	104	4.2%	101	4.3%	205	4.2%
Iritis:						
cause unknown	74		85		159	
cause known	58		34		92	
	132	5.4%	119	5.1%	251	5.3%
Choroiditis:						
cause unknown	33		20		53	
cause known	59		18		77	
	92	3.7%	38	1.6%	130	2.8%
Myopia	5	—	3	—	8	—
Senile macular lesions	5	—	—	—	5	—
Retinal detachment	3	—	2	—	5	—
Diabetic retinopathy	1	—	1	—	2	—
Vascular retinopathy	—	—	1	—	1	—
Optic atrophy :						
Syphilitic	138		70		208	
Other (ecxl. genetic)	67		56		123	
	205	8.4%	126	5.4%	331	6.9%
Normal globe (presumed intracranial cause)	5	—	4	—	9	—
Additional causes, not mentioned in Sorsby's list:						
Keratomalacia	188	7.7%	119	5.1%	307	6.4%
Trachoma	325	13.3%	442	18.2%	767	16.1%
Conj. gonorrhoica:						
neonatorum	11		1		12	
non-neonatorum	151		199		350	
	162	8.2%	200	8.5%	362	7.4%
All other causes	909	37.3%	664	28.6%	1573	33%
Total	2435	100%	2318	100%	4753	100%

Table C 3. Causes of blindness at different ages. Same cause in both eyes. Undaan 1958—62. Visual acuity less than $1^1/_2/60$. (Compare Table 9, Sorsby, 1966, p. 13).

Cause	0—17 yrs				16—41 yrs				Over 40 yrs.			
	M	%	F	%	M	%	F	%	M	%	F	%
Congenital malformations	62	16%	51	17.7%	18	1.6%	9	0.8%	2	0.2%	—	—
(catar. cong.)	(29)		(23)		(4)		(1)		(—)		(—)	
Congenit. syphilis	4	1%	3	1%	—	—	—	—	—	—	—	—
Retinoblastoma	2	0.5%	5	1.7%	—	—	—	—	—	—	—	—
Conj. gonorrh. neonatorum	10	2.5%	1	0.3%	1	0.1%	—	—	—	—	—	—
Idem, non-neonatorum	11	2.8%	16	5.5%	121	10.8%	143	13.4%	19	2 %	40	4.1%
Keratomalacia	188	48.7%	115	40%	—	—	—	—	—	—	—	—
Retinitis pigmentosa	—	—	—	—	14	1.2%	10	0.9%	12	1.3%	7	0.7%
Corneal disease, Wr +	—	—	—	—	26	2.3%	28	2.6%	19	2%	11	1.1%
Idem, other	66	17.1%	55	19%	415	37%	310	29%	222	23.8%	161	16.8%
Uveitis, Wr +	1	0.25%	3	1%	56	5%	28	2.6%	51	5.4%	13	1.3%
Idem, other	8	2%	7	2.4%	61	5.4%	66	6.1%	47	5%	42	4.3%
Ophthalmia sympathica	1	0.25%	—	—	1	0.1%	—		—	—	1	0.1%
Glaucoma	1	0.25%	3	1%	41	3.6%	109	10.2%	155	16.6%	289	30%
Senile cataract	—	—	—	—	11	0.9%	19	1.7%	93	10%	82	8.5%
Trachoma	6	1.6%	2	0.6%	122	10.8%	186	17.3%	197	21.1%	254	26.3%
Optic atrophy, syphilitic	2	0.5%	—	—	74	6.6%	46	4.3%	62	6.6%	24	2.4%
Idem, other	19	2%	18	6.2%	34	3.2%	3	0.2%	14	1.5%	11	1.1%
All other causes	5	1.3%	9	3.1%	121	10.4%	205	18.6%	37	3.9%	27	2.8%
Total	386	100%	288	100%	1119	100%	1067	100%	930	100%	963	100%

Note: Corneal disease: excl. conj. gonorrhoica, Keratomalacia, Trachoma and congenital.

Table C 4. Causes of blindness, Undaan 1958-62, by sex. Adjusted female incidence against actual male incidence. According to the rate male/female in the general population, the adjustment has been worked out as follows: rate male/female for 0—17 year age group: 1/1.05; for 16-41 year age group: 1/1.34 ; for over 40 age group: 1/1.25. Same cause of blindness in both eyes. Visual acuity less than $1^{1}/_{2}/60$. (Compare Table 10, Sorsby, 1966, p. 16)

Cause	0—17 yrs.		16—41 yrs.		Over 40 yrs.	
	M	F	M	F	M	F
Congenital malformations	62	49	18	7	2	—
(congen. catar.)	(29)	(22)	(4)	(<1)	(—)	(—)
Congenital syphilis	4	3	—	—	—	<1
Retinoblastoma	2	5	—	—	—	—
Conj. gonorrh. neonatorum	10	1	1	—	—	—
Idem, non-neonatorum	11	14	121	107	19	32
Keratomalacia	188	110	—	3	—	—
Retinitis pigmentosa	—	—	14	7	12	5
Corneal disease, Wr +	—	—	26	21	19	9
Idem, Other	66	52	415	231	222	124
Uveitis, Wr +	1	3	56	21	51	10
Idem, Other	8	7	61	49	47	34
Ophthalmia sympathica	1	—	1	—	—	<1
Glaucoma	1	3	41	81	155	231
Senile cataract	—	—	11	14	93	66
Trachoma	6	2	122	139	197	203
Optic atrophy, syphilitic	2	—	74	34	62	19
Idem, other	19	17	34	2	14	9
All other causes	5	8	121	153	37	22

Note: Corneal disease: excluding Trachoma, Keratomalacia, conj. gonorrhoica, congenital.

Sorsby : ”Incidence and Causes of Blindness in England and Wales, 1955 — 1962” 1966, Table 9, p. 13. Major causes of blindness in different age groups by sex, after adjustment of age groups. Age at registration. 1955 — 1962, (except oldest age groups, 1955 — 1960 only).

Cause	0—14 years				15—59 years				60 years and over			
	M	F	P	%	M	F	P	%	M	F	P	%
Congenital defects :												
Cataract	122	95	217	13.9%	226	162	388	4.2%	67	71	137	0.2%
Optic atrophy	163	94	257	16.2%	75	48	123	1.3%	14	4	18	0.03%
Other structural defects, incl. nystagmus	250	162	412	26%	19	114	243	2.6%	32	47	79	0.16%
Ophthalmia neonatorum	—	1	1	...	19	21	40	0.4%	4	25	29	0.05%
Interstitial keratitis	—	—	—	—	41	91	132	1.4%	41	80	121	0.24%
Retrolental fibroplasia	81	90	171	10.8%	—	—	—	—	—	—	—	—
Retinoblastoma	30	26	56	3.5%	—	—	—	—	—	—	—	—
Retinal aplasia	16	11	27	1.6%	—	—	—	—	—	—	—	—
Retinitis pigmentosa & allied conditions	17	9	26	1.7%	480	332	812	8.9%	155	180	335	0 67%
Macular dystrophy	8	4	12	0.7%	41	42	83	0.8%	1	11	12	0.02%
Retinal detachment, myopic	7	1	8	0.5%	111	52	163	1.7%	39	66	105	0.21%
Idem, unspecified	6	7	13	0.7%	158	83	241	2.6%	108	99	207	0.4%
Myopic chorioretinal atrophy	16	5	21	1.4%	559	730	1289	14.1%	1118	2693	4011	8%
Glaucoma	—	—	—	—	271	257	528	5.7%	2897	3977	6874	13.8%
Senile cataract	—	—	—	—	182	188	370	4%	3779	9015	15994	32.2%
Senile macular lesions	—	—	—	—	23	36	59	0.6%	5704	9844	15548	31.3%
Diabetic retinopathy	—	1	1	...	461	666	1127	12.3%	588	2617	3105	6.7%
Affections of globe as a whole	—	—	—	—	12	25	37	0.4%	10	65	75	0.15%
Hypertensive & vascular retinopathy	—	—	—	—	91	91	182	2 %	656	1047	1703	3.4%
Iritis & iridocyclitis of undetermined origin	11	10	21	1.4%	182	257	439	4.8%	241	596	837	1.6%
Optic atrophy, known or presumed acquired	131	105	236	15%	781	533	1314	14.4%	643	662	1305	2.6%

Cause	0—14 years				15—59 years				60 years and over			
	M	F	P	%	M	F	P	%	M	F	P	%
Corneal lesions, unspecified	7	3	10	0.6%	117	176	293	3.2%	300	590	890	1.6%
Trauma	10	6	16	1%	145	20	165	1.8%	76	26	102	0.2%
All other causes	43	31	74	4.6%	564	516	1080	11.8%	604	831	1435	2.8%
			1579	100%			9108	100 %			49622	100%

Note: In Sorsby's original Table 9 there are 7 age groups; to make his data more comparable to those of the present study, where a more refined division in age groups was impracticable, Sorsby's figures have been arranged anew in 3 age groups, and percentages have been calculated anew also for these 3 age groups, as is shown above.

Table C 5. Blindness from the same cause in both eyes, Undaan 1/8'66-1/8'67. Visual acuity less than $1^{1}/_{2}/60$. Same criteria for cataract as in 1958-62. Compare Table A 11, 12, 18, 24, 30, 36 (and Table 7, Sorsby, 1966, p.10 and 11).

Classification by **site**.

Site	0—17 yrs		16—41 yrs		Over 40 years		Total	
Eyeball	22	18.3%	49	22.7%	80	35.1%	151	26.8%
Conjunctiva	—	—	8	3.7%	2	0.8%	10	1.7%
Cornea	72	60 %	82	38.1%	70	30.8%	224	39.7%
Lens	6	5 %	11	5.1%	39	17.1%	56	10. %
Uveal tract	3	2.4%	16	7.4%	13	5.7%	32	5.6%
Retina	4	3.3%	5	2.3%	10	4.3%	19	3.3%
Optic nerve, optic pathways, visual centres	6	5 %	39	18.2%	12	5.2%	57	10.1%
Other	7	5.8%	6	2.7%	1	0.4%	14	2.4%
Total	120	100%	216	100%	227	100%	563	100%
Classification by **etiology.**								
Infectious *)	7	5.8%	75	34.9%	69	30.3%	151	26.8%
Trauma	1	0.8%	6	2.7%	4	1.7%	11	1.9%
Systemic diseases	76	63.3%	2	0.9%	4	1.7%	82	14.5%
Prenatal influences	24	20 %	7	3.2%	3	1.3%	34	6 %
Undetermined	12	10 %	126	58.1%	147	64.7%	285	50.6%

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*) **Note :** infectious : excl. transmitted maternal infections.

Tabel C 6. Blindness from same cause in both eyes. Undaan, 1/8'66-1/8'67. Visual acuity **less than** 1/10. Same criteria for cataract as in 1958-62. Compare Tables A 37, 38, 39, 40, 41 and 42 (and Table 7 from Sorsby, 1966, p. 10 and 11)

Classification by **site**.

Site	0—17 yrs		16—41 yrs		Over 40 yrs		Total	
Eyeball	26	17.3%	62	16.8%	87	20%	175	18.4%
Conjunctiva	1	0.6%	14	3.8%	4	0.9%	9	0.9%
Cornea	101	67.3%	196	53.4%	134	30.9%	431	45.3%
Lens	10	6.6%	17	4.6%	150	34.6%	177	18.6%
Uveal tract	3	2%	23	6.2%	26	6%	52	5.4%
Retina	6	4%	7	1.9%	15	3.5%	28	2.9%
Optic nerve, optic pathways, visual centres	6	4%	40	10.9%	12	2.7%	58	6.1%
Other	7	4.6%	8	2.2%	5	1.1%	20	2%
Total	150	100%	367	100%	433	100%	950	100%
Classification by **etiology.**								
Infectious *)	17	11.3%	143	38.9%	117	27%	277	29.1%
Trauma	1	0.6%	8	2.2%	7	1.6%	16	1.6%
Systemic disease	84	56%	3	0.8%	8	1.8%	95	10%
Prenatal influences	29	19.3%	10	2.7%	3	0.6%	42	4.4%
Undetermined	19	12.6%	203	55.3%	298	68.8%	420	44.2%

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*) **Note :** infectious : excluding transmitted maternal infections.

Table C 7. Clinical entities, responsible for blindness. Same cause in both eyes. Undaan 1/8'66-1/8'67. Visual acuity less than $1^1/_2/60$. All ages by sex. Same criteria for cataract as in 1958-62. Compare Tables A 11, 12, 18, 24, 30, 36 and Table 8, Sorsby 1966, p. 12.

Clinical entity	Males		Female		Total	
Congenital defects :						
Globe as a whole	6		11		17	
Cataract	5		1		6	
Retinoblastoma	—		1		1	
	11	3.8%	13	4.5%	24	4.2%
Abiotrophic defects :						
Retinitis pigmentosa	5		1		6	
Macular dystrophy	4		—		4	
	9	3.1%	1	0.3%	10	1.7%
Glaucoma	27	9.6%	74	26.2%	101	17.9%
Interstitial keratitis	3	1%	—	—	3	0.5%
Senile cataract	28	9.9%	20	7%	48	8.5%
Iritis, unknown cause	6		3		9	
Idem, known cause	3		1		4	
	9	3.1%	4	1.3%	13	2.3%
Choroiditis, unknown cause	10		2		12	
Idem, known cause	2		2		4	
	12	4.2%	4	1.3%	16	2.8%
Myopia	3	1%	5	1.7%	8	1.4%
Senile macular lesion	4	1.4%	—	—	4	0.7%
Retinal detachment	5	1.7%	1	0.3%	6	1%
Optic atrophy, luetic	20	7.1%	7	2.4%	27	4.7%
Idem, other cause, excl. genetic	8	2.8%	22	7.8%	30	5.2%
Normal globe (presumed intracranial cause)	4	1.4%	6	2.1%	10	1.7%
Additional causes, not mentioned in Sorsby's list :						
Keratomalacia	38	13.6%	30	10.6%	68	12%
Conj. gonorrhoica, nonneonatorum	22	7.8%	26	9.2%	48	8.6%
Trachoma	20	7.1%	35	12.4%	55	9.9%
All other causes	58	20.6%	34	12%	92	17.5%
Total	281	100%	282	100 %	563	100%

Table C 8. Causes of blindness, both eyes same cause. Age groups by sex. Undaan 1/8'66—1/8'67. Visual acuity less than $1^1/_2/60$. Same criteria for cataract as in 1958—62. Compare Tables A 11, 12, 18, 24, 30 and 36, and Sorsby, Table 9, 1966. p. 13.

Cause	0—17 yrs				16—41 yrs				Over 40 yrs			
	M		F		M		F		M		F	
Congenital malformations	9	15.2%	9	14.7%	2	1.9%	—	—	—	—	—	—
(congen. catar.)	(5)		(1)		—	—	—	—	—	—	—	—
Retinoblastoma	—	—	1	1.6%	—	—	—	—	—	—	—	—
Conj. gonorrh. non-neonatorum	1	1.6%	2	3.2%	14	13.3%	18	16.3%	5	4.3%	6	5.4%
Keratomalacia	38	64.4%	30	49%	—	—	—	—	—	—	—	—
Retinit. pigm.	1	1.6%	—	—	1	0.9%	1	0.9%	3	2.5%	—	—
Corneal disease Wr +	—	—	—	—	1	0.9%	—	—	2	1.7%	—	—
Idem, Wr —	2	3.2%	1	1.6%	23	21.9%	22	20%	22	19%	10	9%
Uveitis, Wr +	—	—	—	—	1	0.9%	1	0.9%	4	3.4%	2	1.8%
Idem, other	1	1.6%	1	1.6%	8	7.5%	4	2.6%	6	5.1%	—	—
Ophth. sympath.	—	—	—	—	1	0.9%	1	0.9%	—	—	1	0.9%
Glaucoma	1	1.6%	3	4.8%	6	5.7%	25	22.7%	22	19%	49	44%
Senile cataract	—	—	—	—	7	6.6%	3	2.7%	21	18%	12	10.8%
Trachoma	—	—	1	1.6%	6	5.7%	8	7.2%	14	12%	21	19%
Optic atrophy, luetic	—	—	—	—	14	13.2%	4	3.6%	6	5%	3	2.7%
Idem, other	1	1.6%	5	8%	7	6.6%	14	12.7%	—	—	3	2.7%
All other causes	5	8.4%	8	13%	15	14.3%	9	8%	11	9.4%	4	3.6%
Total	59	100%	61	100%	106	100%	110	100%	116	100%	111	100%

Table C 9. Clinical entities, responsible for blindness, same cause in both eyes. Undaan, 1/8'66-1/8'67. Visual acuity **less than 1/10.** All ages by sex. Same criteria for cataract as in 1958-62. Compare Tables A 37-42 and Sorsby, Table 8, 1966, p. 12

Clinical entity	Male		Female		Total	
Congenital defects :						
Globe as a whole	6		7		13	
Cataract	9		2		11	
Uveal malformations	1		—		1	
Retinoblastoma	—		1		1	
	16	3.3%	10	2 %	26	2.6%
Abiotrophic defects :						
Retinitis pigmentosa	5		1		6	
Macular dystrophy	—		1		1	
	5	1 %	2	0.4%	7	0.7%
Glaucoma	32	6.7%	78	16.3%	110	11.5%
Interstitial keratit.	3	0.6%	—	—	3	0.3%
Senile cataract	92	19.5%	72	15 %	164	17 %
Iritis, unknown cause	11	2.3%	8	1.6%	19	2 %
Idem, known cause	6	1.2%	4	0.8%	10	1 %
Choroiditis, unknown cause	10	2.1%	3	0.6%	13	1.3%
Idem, known cause	2	0.4%	3	0.6%	5	0.5%
Myopia	12	2.5%	9	1.8%	21	2.1%
Senile macular lesions	7	1.4%	2	0.4%	9	0.9%
Retinal detachment	6	1.2%	1	0.2%	7	0.7%
Optic atrophy, luetic	20	4 %	7	1.4%	27	2.7%
Idem, other cause	8	1.7%	23	4.8%	31	3.1%
Normal globe, presumed intracranial cause	6	1.2%	8	1.6%	14	1.4%
Additional entities, not listed by Sorsby :						
Keratomalacia	42	8.8%	34	7.1%	76	8 %
Conj. gonorrh. non-neonatorum	35	7.3%	30	6 %	65	6.8%
Trachoma	54	11.4%	95	19.9%	149	15.8%
All other causes	115	24.3%	87	18.4%	192	20.2%
Total	472	100%	477	100%	949	100%

Table C 10. Causes of blindness, same cause in both eyes. Undaan 1/8'66-1/8'67. Visual acuity less than $1^1/_2/60$. Same criteria for cataract as in 1958-62. Adjusted female incidence against actual male incidence. Rate of adjustment for 1966 : 0-17 yrs. 1.18: 16-41 yrs.: 1.25; over 40 yrs: 1.08. Compare Tables A 11, 12, 18, 24, 30, 36 and Table 10, Sorsby, 1966, p. 16.

Cause	0—17 yrs		16—41 yrs		over 40 yrs.	
	M	F	M	F	M	F
Congenital malformations	9	7	2	—	—	—
(congen. cataract)	(5)	(1)	—	—	—	—
Retinoblastoma	—	<1	—	—	—	—
Conj. gonorrh. non-neonat.	1	2	14	14	5	6
Keratomalacia	38	25	—	—	—	—
Retinitis pigmentosa	1	—	1	<1	3	—
Corneal disease, Wr +	—	—	1	—	2	—
Idem, other cause	2	<1	23	18	22	9
Uveitis, Wr +	—	—	1	<1	4	2
Idem, other cause	1	<1	8	3	6	—
Ophth. sympathica	—	—	1	<1	—	<1
Glaucoma	1	3	6	20	22	45
Senile cataract	—	—	7	2	21	11
Trachoma	—	<1	6	6	14	19
Optic atrophy, luetic	—	—	14	3	6	3
Idem, other cause	1	4	7	12	—	3
All other causes	5	7	15	7	11	4
Total	59	(52)	106	(88)	135	(110)

Note : Corneal disease : excluding Trachoma, Keratomalacia, conj. gonorrhoica, cangenital.

Table C 11. Causes of blindness, same cause in both eyes. Age groups by sex. Undaan, 1/8'66—1/8'67. Visual acuity **less than 1/10.** Same criteria for cataract as in 1958—62. Compare Tables A 37—42 and Table 9. Sorsby 1966, p. 13.

Cause	0—17 yrs				16—41 yrs				over 40 yrs			
	M		F		M		F		M		F	
Congenital malformations	14	19.1%	10	13%	3	1.3%	2	1%	—	—	—	—
(Congen. catar.)	(9)		(1)		(—)		(1)		—		—	
Retinoblastoma	—	—	1	1.3%	—	—	—	—	—	—	—	—
Conj. gonorrh. non-neonatorum	2	2.7%	3	3.8%	26	14.7%	20	10.5%	8	3.6%	7	3.3%
Keratomalacia	41	56%	34	44%	—	—	—	—	—	—	—	—
Retinit. pigment.	1	1.3%	—	—	1	0.6%	1	0.5%	3	1.3%	—	—
Corneal disease, Wr +	—	—	—	—	1	0.6%	—	—	2	0.9%	—	—
Idem, other	5	6.8%	5	6.5%	48	27%	56	29.4%	33	14.8%	22	10.4%
Uveitis, Wr +	—	—	—	—	2	1.1%	2	1%	6	2.7%	4	1.9%
Idem, other	2	2.9%	1	1.3%	12	6.7%	5	2.6%	9	4%	6	2.8%
Ophth. sympathica	—	—	—	—	1	0.6%	1	0.5%	—	—	1	0.5%
Glaucoma	1	1.3%	3	3.8%	7	4%	25	13%	24	10.8%	50	23.8%
Senile cataract	—	—	—	—	11	6.2%	5	2.6%	78	35%	61	29%
Trachoma	1	1.3%	5	6.5%	22	12.4%	43	22.6%	31	14%	36	17%
Optic atrophy, Luetic	—	—	—	—	14	7.9%	4	2.1%	6	2.7%	3	1.4%
Idem, other	1	1.3%	5	6.5%	8	4.5%	15	7.8%	—	—	3	1.4%
All other causes	5	6.8%	10	13%	31	17.5%	11	5.8%	22	9.9%	10	4.7%
Total	73	100%	77	100%	177	100%	190	100%	222	100%	210	100%

Note: Corneal disease: excl. those noted under: Trachoma, Keratomalacia, gonorrhoica conjunctivitis, genetic.

Vable C 12. Causes of blindness, same cause in both eyes. Undaan 1/8'66-1/8'67. feisual acuity **less than 1/10.** Same criteria for cataract as in 1958-62. Adjusted 1.male incidence against actual male incidence. Adjustment rate for 1966 ; 0-17 yrs, S 18 ; 16-41 yrs : 1. 25 ; over 40 yrs : 1. 08. Compare Tables A 37-42 and Table 10. Torsby, 1966, p. 16.

Cause	0—17 yrs		16—41 yrs		over 40 yrs	
	M	F	M	F	M	F
Congenital malformation	14	8	3	2	—	—
(congen. cataract)	(9)	(<1)	—	(<1)	—	—
Retinoblastoma	—	<1	—	—	—	—
Conj. gonorrh. non-neonat.	2	2	26	16	8	6
Keratomalacia	41	30	—	—	—	—
Retinit. pigmentosa	1	—	1	<1	3	—
Corneal disease, Wr +	—	—	1	—	2	—
Idem, other	5	4	48	45	33	20
Uveitis, Wr +	—	—	2	2	6	4
Idem, other	2	<1	12	4	9	6
Ophth. sympathica	—	—	1	<1	—	<1
Glaucoma	1	2	7	20	24	47
Senile cataract	—	—	11	4	78	61
Trachoma	1	4	22	34	31	33
Optic atrophy, Wr +	—	—	14	3	6	3
Idem, other	1	4	8	12	—	3
All other causes	5	8	21	9	22	9
Total	73	(64)	177	(153)	222	(193)

Note: corneal disease excl. Trachoma, Keratomalacia, conj. gonorrhoica, genetic.

Table C 13. Causes of blindness, same cause in both eyes. Undaan 1/8'66-1/8'67. Visual acuity less than $1^1/_2/60$. Including all cataract cases. Compare Table 7, Sorsby, 1966, p. 10 and 11.

Classification by **site.**

Site	0—17 yrs		16—41 yrs		over 40 yrs		Total	
Eyeball	22	18.3%	49	22.7%	80	30.7%	151	25.5%
Conjunctiva	—	—	8	3.7%	2	0.7%	10	1.6%
Cornea	72	60%	82	38%	70	26.9%	224	37.6%
Lens	6	5%	11	5%	72	27.6%	89	14.9%
Uveal tract	3	2.4%	16	7.4%	13	5 %	32	5.2%
Retina	4	3.3%	5	2.3%	10	3.8%	19	3.1%
Optic nerve, optic pathways, visual centre	6	5 %	39	18.1%	12	3.2%	57	9.3%
Other	7	5.8%	6	2.7%	1	0.3%	14	2.3%
Total	120	100%	216	100%	260	100%	596	100%
Classification by **etiology.**								
Infectious *)	7	5.8%	75	35 %	69	26.5%	151	25.5%
Trauma	1	0.8%	6	2.7%	4	1.4%	11	1.9%
Systemic disease	76	63.3%	2	0.9%	4	1.4%	82	13.7%
Prenatal influence	24	20 %	7	3.2%	3	1 %	34	5.7%
Undetermined	12	10 %	126	58 %	180	69.2%	318	53.2%

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*) **Note :** Infectious, excl. transmitted maternal infections.

Table C 14. Causes of blindness, same cause in both eyes. Undaan 1/8'66-1/8'67. Visual acuity **less than 1/10.** Including all cataract cases. Compare Table 7, Sorsby, 1966, p. 10 and 11.

Classification by **site.**

Site	0—17 yrs		16—41 yrs		Over 40 yrs		Total	
Eyeball	26	17.3%	62	16.2%	87	17.9%	175	1.7%
Conjunctiva	1	0.6%	14	3.6%	4	0.6%	9	0.7%
Cornea	101	67.3%	196	51%	134	27.7%	431	42.1%
Lens	10	6.6%	34	8.8%	207	41.6%	251	24.5%
Uveal tract	3	2%	23	6%	26	5.3%	52	5%
Retina	6	4%	7	1.8%	15	3%	28	2.7%
Optic nerve, optic pathways, visual centre	6	4%	40	10.5%	12	2.4%	58	5.4%
Other	7	4.6%	8	2%	5	1%	20	1.9%
Total	150	100 %	384	100%	490	100%	1024	100 %
Classification by **etiology**								
Infectious *)	17	11.3%	143	37%	117	23.8%	277	27%
Trauma	1	0.6%	8	2%	7	1.4%	16	1.5%
Systemic diseases	84	56%	3	0.7%	8	1.6%	95	9.2%
Prenatal influences	29	19.3%	10	2.6%	3	0.6%	42	4%
Undetermined	19	12.6%	220	57%	355	74.2%	584	57%

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*) **Note:** infectious, excl. transmitted maternal infections.

Table C 15. Clinical entities, responsible for blindness, same cause in both eyes. Undaan, 1/8'66-1/8'67. Visual acuity less than $1^{1}/_{2}/60$. All ages by sex. Compare Table 8, Sorsby, 1966, p. 12.

Clinical entity	Male		Female		Total	
Congenital defects :						
Globe as a whole	6		11		17	
Cataract	5		1		6	
Retinoblastoma	—		1		1	
	11	3.6%	13	4.3%	24	3.9%
Abiotrophic defects :						
Retinitis pigmentosa	5		1		6	
Macular dystrophy	4		—		4	
	9	2.9%	1	0.3%	10	1.7%
Glaucoma	27	9 %	74	25 %	101	16.9%
Interstitial keratitis	3	1 %	—	—	3	0.5%
Senile cataract	47	15.7%	34	11.3%	81	13.6%
Iritis, unknown cause	6		3		9	
Idem, known cause	3		1		4	
	9	3 %	4	1.2%	13	2.2%
Choroiditis, unknown cause	10		2		12	
Idem, known cause	2		2		4	
	12	4 %	4	1.2%	16	2.6%
Myopia	3	1 %	5	1.5%	8	1.3%
Senile macular lesions	4	1.3%	—	—	4	0.7%
Retinal detachment	5	1.6%	1	0.3%	6	1 %
Optic atrophy, luetic	20	6.6%	7	2.1%	27	4.5%
Idem, other cause	8	2.6%	22	7.4%	30	5 %
Normal globe, presumed intracranial cause	4	1.3%	6	1.8%	10	1.7%
Additional entities, not mentioned in in Sorsby's list :						
Keratomalacia	38	12.7%	30	10 %	68	11.3%
Conj. gonorrhoica, non-neonatorum	22	7.3%	26	8.7%	48	8 %
Trachoma	20	6.6%	35	11.6%	55	9.2%
All other causes	58	19 %	34	11.3%	92	15.4%
Total	300	100%	296	100%	596	100%

Table C 16. Causes of blindness, both eyes same cause. In age groups by sex. Undaan, 1/8'66—1/8'67. Visual acuity less than $1^{1}/_{2}/60$. Compare Table 9, Sorsby, 1966, p. 13. Including all cataract cases.

Cause	0—17 yrs				16—41 yrs				over 50 yrs			
	M		F		M		F		M		F	
Congenital malformations	9	15.2%	9	14.7%	2	1.9%	—	—	—	—	—	—
(congen. catar.)	(5)		(1)		—	—	—	—	—	—	—	—
Retinoblastoma	—	—	1	1.6%	—	—	—	—	—	—	—	—
Conj. gonorrh. non-neonatorum	1	1.6%	2	3.2%	14	13.2%	18	16.3%	5	3.6%	6	4.8%
Keratomalacia	38	64.4%	30	49%	—	—	—	—	—	—	—	—
Retinit. pigm.	1	1.6%	—	—	1	0.9%	1	0.9%	3	2.2%	—	—
Corneal disasease, Wr +	—	—	—	—	1	0.9%	—	—	2	1.4%	—	—
Idem, other	2	3.2%	1	1.6%	23	21.9%	22	20%	22	16.3%	10	8%
Uveitis, Wr +	—	—	—	—	1	0.9%	1	0.9%	4	2.9%	2	1.6%
Idem, Wr —	1	1.6%	1	1.6%	8	7.5%	4	3.6%	6	4.4%	—	—
Ophth. sympath.	—	—	—	—	1	0.9%	1	0.9%	—	—	1	0.8%
Glaucoma	1	1.6%	3	4.8%	6	5.7%	25	22.7%	22	16.3%	49	39.2%
Senile cataract	—	—	—	—	7	6.6%	3	2.7%	40	29.5%	31	24.8%
Trachoma	—	—	1	1.6%	6	5.7%	8	7.2%	14	10.3%	26	10.8%
Optic atrophy, syphilitic	—	—	—	—	14	13.2%	4	3.6%	6	4.4%	3	2.4%
Idem, other	1	1.6%	5	8%	7	6.6%	14	12.7%	—	—	3	2.4%
All other causes	5	8.4%	8	13%	15	14.3%	9	8.1%	11	8.1%	4	3.2%
Total	59	100 %	61	100 %	106	100 %	110	100 %	135	100 %	125	100 %

Note : corneal disease : excl. those cases caused by Trachoma, Keratomalacia, conj. gonorrhoica, genetic.

Table C 17. Clinical entities responsible for blindness, same cause in both eyes. Undaan, 1/8'66-1/8'67 Visual acuity **less than 1/10.** All cataract cases included. All ages by sex. Compare Table 8, Sorsby, 1966, p. 12.

Clinical entity.	Males		Females		Total	
Congenital defects:						
Globe as a whole	6		7		13	
Cataract	9		2		11	
Uveal malformation	1		—		1	
Retinoblastoma	—		1		1	
	16	3.1%	10	1.9%	26	2.5%
Abiotrophic defects:						
Retinitis pigment.	5		1		6	
Macular dystrophy	—		1		1	
	5	0.9%	2	0.4%	7	0.7%
Glaucoma	32	6.2%	78	15.3%	110	10.6%
Interstitial keratit.	3	0.6%	—	—	3	0.3%
Senile cataract	135	26.2%	104	20.4%	239	23.3%
Iritis, unknown cause	11	2.1%	8	1.6%	19	1.8%
Idem, known cause	6	1.1%	4	0.8%	10	1 %
Choroiditis, unknown cause	10	1.7%	3	0.6%	13	1.1%
Idem, known cause	2	0.2%	3	0.6%	5	0.5%
Myopia	12	2.5%	9	1.4%	21	2 %
Senile macular lesion	7	1.5%	2	0.4%	9	0.8%
Retinal detachment	6	1.1%	1	0.2%	7	0.6%
Optic atrophy, luetic	20	3.8%	7	1.4%	27	2.5%
Idem, other cause	8	1.5%	23	4.6%	31	3 %
Normal globe (presumed intracranial cause)	6	1.1%	8	1.6%	14	1.3%
Additional entities, not mentioned in Sorsby's list:						
Keratomalacia	42	8.3%	34	6.6%	76	7.4%
Conj. gonorrh. non-neonatorum	35	6.7%	30	5.9%	65	5.3%
Trachoma	54	10.4%	95	18.7%	149	14.5%
All other causes	115	22.3%	87	17.1%	192	18.7%
Total	515	100%	508	100%	1023	100%

Table C 18. Causes of blindness, both eyes same cause. Undaan, 1/8'66—1/8'67. Visual acuity **less than 1/10.** In age groups by sex. All cataract cases included. Compare Table, 9, Sorsby, 1966, p. 13.

Cause	0—17 yrs				16—41 yrs				Over 40 yrs			
	M		F		M		F		M		F	
Congenital malformations	14	19.1%	10	13 %	3	1.5%	2	1 %	—	—	—	—
(Congen. catar.)	(9)		(1)		(—)		(1)					
Retinoblastoma	—	—	1	1.3%	—	—	—	—		—	—	—
Conj. gonorrh. non-neonatorum	2	2.7%	3	3.8%	26	13.6%	20	10.3%	8	3.1%	7	2.9%
Keratomalacia	41	56%	34	44%	—	—	—	—	—	—	—	—
Retinitis pigment.	1	1.3%	—	—	1	0.5%	1	0.5%	3	1.1%	—	—
Corneal disease, Wr +	—	—	—	—	1	0.5%	—	—	2	0.7%	—	—
Idem, other	5	6.8%	5	6.5%	48	25.2%	56	29%	33	13%	22	9.2%
Uveitis, Wr +	—	—	—	—	2	1%	2	1%	6	2.3%	4	1.6%
Idem, other	2	2.9%	1	1.3%	12	6.3%	5	2.5%	9	3.5%	6	2.4%
Ophth. sympathica	—	—	—	—	1	0.5%	1	0.5%	—	—	1	0.4%
Glaucoma	1	1.3%	3	3.8%	7	3.6%	25	12.8%	24	9.5%	50	21%
Senile cataract	—	—	—	—	24	12.6%	9	4.6%	108	42.8%	95	40%
Trachoma	1	1.3%	5	6.5%	22	11.5%	43	22.2%	31	12.3%	36	14.8%
Optic atrophy, luetic	—	—	—	—	14	7.3%	4	2%	6	2.3%	3	1.2%
Idem, other	1	1.3%	5	6.5%	8	4.2%	15	7.7%	—	—	3	1.2%
All other causes	5	6.8%	10	13%	31	16.3%	11	5.6%	22	8.7%	10	4.2%
Total	73	100%	77	100%	190	100%	194	100%	252	100%	237	100%

Note: corneal disease, excl. Trachoma, Keratomalacia, conj. gonorrhoica, genetic.

Table C 19. Causes of blindness, same cause in both eyes. Undaan 1/8'66-1/8'67. Visual acuity less than $1^1/_2/60$. Adjusted female incidence against actual male incidence. Rate of adjustment for 1966: 0—17 yrs. 1.18; 61—41 yrs: 1.25; over 40 yrs: 1.08. All cataract cases included. Compare Table 10 Sorsby, 1966, p. 16.

Cause	0—17 yrs		16—41 yrs		Over 40 yrs	
	M	F	M	F	M	F
Congenital malformations	9	7	2	—	—	—
(Congen. catar.)	(5)	(1)	—	—	—	—
Retinoblastoma	—	<1	—	—	—	—
Conj. gonorrhoica non-neonatorum	1	2	14	14	5	6
Keratomalacia	38	25	—	—	—	—
Retinitis pigmentosa	1	—	1	<1	3	—
Corneal disease, Wr +	—	—	1	—	2	—
Idem, other	2	<1	23	18	22	9
Uveitis, Wr +	—	—	1	<1	4	2
Idem, other	1	<1	8	3	6	—
Ophth. sympathica	—	—	1	<1	—	<1
Glaucoma	1	3	6	20	22	45
Senile cataract	—	—	7	2	40	29
Trachoma	—	<1	6	6	14	24
Optic atrophy, luetic	—	—	14	3	6	3
Idem, other	1	4	7	12	—	3
All other causes	5	7	15	7	11	4
Total	59	(52)	106	(88)	135	(126)

Table C 20. Causes of blindness, same cause in both eyes, Undaan 1/8'66-1/8'67. Visual acuity **less than 1/10.** All cataract cases included. Adjusted female incidence against actual male incidence. Rate of adjustment see table C 19. Compare Table 10, Sorsby, 1966, p. 16.

Cause	0—17 yrs		16—41 yrs		over 40 yrs	
	M	F	M	F	M	F
Congenital malformations	14	8	3	2	—	—
(Congen. catar.)	(9)	(<1)	—	(<1)	—	—
Retinoblastoma	—	<1	—	—	—	—
Conj. gonorrhoica non-neonatorum	2	2	26	16	8	6
Keratomalacia	41	30	—	—	—	—
Retinit. pigmentosa	1	—	1	<1	3	—
Corneal disease, Wr +	—	—	1	—	2	—
Idem, other	5	4	48	45	33	20
Uveitis, Wr +	—	—	2	2	6	4
Idem, other cause	2	<1	12	4	9	6
Ophth. sympathica	—	—	1	<1	—	<1
Glaucoma	1	2	7	20	24	47
Senile cataract	—	—	24	7	108	87
Trachoma	1	4	22	34	31	33
Optic atrophy, luetic	—	—	14	3	6	3
Idem, other	1	4	8	12	—	3
All other causes	5	8	21	9	22	9
Total	73	(64)	190	(156)	252	(219)

Note: corneal disease, excl. Trachoma, Keratomalacia, Conj. gonorrhoica, genetic.

Table A 43. **Monocular** blindness. 94 boys under 17 yrs. of age, seen at Undaan, 1/8'66-1/8'67. Visual acuity less than $1^1/_2/60$. in one eye, more in the other. Classification by site/type and etiology.

	12	13	18	19	20	21	40	45	67	89	91	98	99	Total
110											1			1
130							1						2	3
146										1				1
150									1				2	3
190								1						1
212	3													3
311				1	1							1		3
312						1								1
313		1			1		7						3	12
315	2						1		4			1	2	10
360									21					21
380							1							1
380.5			1						3				2	6
390							1							1
410							10			2	1			13
510												2		2
610											1			1
670											6			6
710													1	1
810							4							4
Total	5	1	1	1	2	1	25	1	29	3	9	4	12	94

Table A 44. **Monocular** blindness. 77 girls, under 17 yrs. of age, seen at Undaan, 1/8'66-1/8'67. Visual acuity less than $1^1/_2/60$ in one eye, more in the other. Classification by site/type and etiology.

	12	13	18	20	21	28	40	67	89	91	98	99	Total
130							1					1	2
147									1				1
150							1	2				1	4
190												1	1
211	1												1
212	1												1
311											1		1
312					1								1
313	1					1	5					3	10
315	2		1					6			1	5	15
316	1			1									2
360								18					18
380.5	1	1						5				2	9
410							2		1				3
670										6			6
710												1	1
810												1	1
Total	7	1	1	1	1	1	9	31	2	6	2	15	77

Table A 45. **Monocular blindness.** 243 men, 16-41 yrs of age, seen at Undaan, 1/8'66-1/8'67. Visual acuity less than $1^1/_2/60$ in one eye, more in the other. Classification by site/type and etiology.

	12	18	19	20	21	40	67	89	91	98	99	E	Ex	Total
110									5					5
122									5					5
130						3				3	4			10
144								1						1
146								1						1
150	1			1		2					7			11
180						1								1
190						2					6			8
212	8													8
311										3				3
313	2	1		6		16				6	27			58
315	5	1	1	1		2	2			2	26			40
316	4										2			6
320											1			1
380						2								2
380.5	1					1					11			13
390						1							1	2
410				1		14		1	18					34
510	1		3			1				11	4			20
560										1	1			2
610										1	1	1		3
640									1					1
660						1					1			2
710			1			1								2
810					1	1				1				3
990											1			1
Total	20	2	5	9	1	48	2	3	29	28	92	1	1	243

E: Eclips

Ex : Exophthalmos.

Table A 46. **Monocular blindness.** 132 women, 16-41 yrs. of age, seen at Undaan, 1/8'66-1/8'67. Visual acuity less than $1^1/_2/60$ in one eye, more in the other. Classification by site/type and etiology.

	12	18	19	20	40	67	89	91	98	99	Total
110								13			13
122								3			3
130					2				1	3	6
150	4		1	2		1				5	13
190	1									6	7
212	7										7
312			2	9	3				1	5	20
315	4	1		1					1	18	25
316	2								3	1	6
380	1										1
380.4					1						1
380.5									1	2	3
410					2		1	10			13
480								1			1
510			1						6		7
610					1				1		2
640									1		1
710										2	2
990										1	1
Total	19	1	4	12	9	1	1	27	15	43	132

Table A 47. **Monocular blindness.** 223 men, over 40 yrs. of age, seen at Undaan, 1/8'66-1/8'67. Visual acuity less than $1^1/_2/60$ in one eye, more in the other. Classification by site/type and etiology.

	12	18	19	20	21	28	40	46	62	89	91	98	99	Ca	Total
110											12				12
122											1				1
130							1						4		5
142										1					1
150	2			2			1	1					6		12
212	2														2
311												1			1
313			4	2		4	9					2	20		41
315		1					1						10		12
316													1		1
380.4				1									1		2
380.5													1		1
410				1			3		2		88				94
510			2		2						1	3	1		9
550												1			1
560												1			1
610			1									1			2
640													1		1
650										1					1
660												1			1
710			4		1							4		1	10
810							1					4	2		7
940												1			1
990												1	3		4
Total	4	1	11	6	3	4	16	1	2	2	102	20	50	1	223

Ca : Carcinoma.

Table A 48. **Monocular blindness.** 177 women, over 40 yrs. of age, seen at Undaan, 1/8'66-1/8'67. Visual acuity less than $1^1/_2/60$ in one eye, more in the other. Classification by site/type and etiology.

	12	19	20	40	62	91	98	99	Ca	Total	Ca : Carcinoma.
110						32				32	
122			1							1	
130								1		1	
150				2				4		6	
190								1		1	
212	1									1	
313			4	1			1	12	1	19	
315			2	1				8		11	
316							1	2		3	
318				1						1	Mooren's ulcer.
380.5								2		2	
390							1			1	
410			1	1	1	71				74	
480						1				1	
510		2					6	1		9	
530							1			1	
560							2			2	
570				1						1	
610					1		2			3	
640								1		1	
710		1					1			2	
810		1						1		2	
990								2		2	
Total	1	4	8	7	2	104	15	35	1	177	

Table C 21. **Monocular blindness.** Causes of blindness in age groups by sex. Undaan, 1/8'66—1/8'67. Visual acuity less than $1\frac{1}{2}/60$ in one eye, more in the other. Classification by site/type and etiology. Compare Tables A 43—48 and Table 9, Sorsby, 1966, p. 13

Cause	0—17 yrs				16—41 yrs				Over 40 yrs			
	M		F		M		F		M		F	
Congenital malformations	3	3.1%	2	2.6%	3	1%	1	0.75%	1	0.4%	—	—
(Congen. catar.)	(2)		(1)		(1)		(1)		—		—	
Retinoblastoma	6	6.4%	6	7.8%	—	—	—	—	—	—	—	—
Ophth. neonator.	—	—	1	1.3%	—	—	—	—	—	—	—	—
Conj. gonorrh. non-neonatorum	5	5.3%	6	7.8%	22	9%	19	14.4%	4	1.8%	1	0.5%
Keratomalacia	29	31%	31	40%	2	0.8%	1	0.75%	—	—	—	—
Retinit. pigment.	—	—	—	—	—	—	—	—	1	0.4%	—	—
Corneal disease, Wr+	1	1%	—	—	1	0.4%	2	1.5%	4	1.8%	—	—
Idem, Wr —	12	12.7%	16	21%	81	33.3%	33	25 %	41	13%	28	16%
Uveitis, Wr +	—	—	—	—	3	1.2%	1	0.75%	2	0.8%	2	1.1%
Idem, other	2	2.1%	—	—	17	7%	6	4.5%	9	4%	10	5.6%
Ophth. sympathica	—	—	—	—	—	—	—	—	—	—	1	0.5%
Glaucoma	1	1%	—	—	5	2%	13	9.8%	12	5.3%	32	18%
Senile cataract	1	1%	—	—	18	7.4%	10	7.5%	90	40%	72	41%
Trachoma	2	2.1%	1	1.3%	9	3.7%	12	9.1%	6	2.6%	8	4.5%
Optic atrophy, luetic	—	—	—	—	1	0.4%	—	—	4	1.8%	1	0.5%
Idem, other	1	1%	1	1.3%	—	—	2	1.5%	6	2.6%	1	0.5%
Trauma	26	27.6%	9	12%	48	20%	9	6.8%	17	7.6%	6	3.4%
All other causes	6	6.4%	4	5.2%	33	13.5%	23	17.4%	26	11.6%	15	8.4%
Total	94	100 %	77	100 %	243	100 %	132	100 %	223	100 %	177	100 %

Note: Corneal disease: excl. conj. gonorrhoica, keratomalacia, Trachoma and congenital.

Table C 22. **Monocular blindness,** Causes of blindness in age groups by sex. Undaan, 1/8'66-1/8'67. Visual acuity less than $1^1/_2/60$ in one eye, more in the other. Adjusted female incidence against actual male incidence. Rate of adjustment for 1966: 0-17 yrs ; 1.18: 16-14 yrs: 1.25 ; over 40 yrs : 1/68. Compare Table 10, Sorsby, 1966, p. 16.

Cause	0-17 yrs		16-41 yrs		Over 40 yrs	
	M	F	M	F	M	F
Congenital malformations	2	3	3	<1	1	—
(congen. catar.)	(2)	(<1)	(1)	(<1)	—	—
Retinoblastoma	6	5	—	—	—	—
Ophth. neonatorum	—	<1	—	—	—	—
Conj. gonorrh. non-neonatorum	5	5	22	16	4	<1
Keratomalacia	29	26	2	<1	—	—
Retinit. pigment.	—	—	—	—	1	—
Corneal disease, Wr +	1	—	1	2	4	—
Idem, other	12	14	81	26	41	26
Uveitis, Wr +	—	—	3	<1	2	2
Idem, other	2	—	17	5	9	9
Ophth. sympathica	—	—	—	—	—	<1
Glaucoma	1	—	5	10	12	30
Senile cataract	1	—	18	8	90	66
Trachoma	2	<1	9	9	6	7
Trauma	26	8	48	7	17	5
Optic atrophy, luetic	—	—	1	—	4	<1
Idem, other	1	<1	—	2	6	<1
All other causes	6	3	33	17	26	14
Total	94	(66)	243	(105)	223	(163)

Table A 49. **Monocular blindness.** 117 boys, under 17 yrs. of age, seen at Undaan, 1/8'66—1/8'67. Visual acuity : **less than 1/10.** in one eye, more in the other. Classification by site/type and etiology.

	12	13	18	19	20	21	40	45	65	67	89	91	98	99	Total
110												1			1
122												1		1	2
130							1							2	3
146											1			2	3
150										1				8	9
190								1						4	5
212	3														3
311				1	1								1		3
312						1									1
313		1			2		8								11
315	2						1			4			1		8
360										23					25
380							1								1
380.5			1							3				2	6
390							1								1
410							11				3	2			16
510													3		3
610												1		1	2
670												6			6
710														1	1
810							5								5
940														1	1
990							1		1					1	3
Total	5	1	1	1	3	1	29	1	1	31	4	11	5	23	117

Table A 50. **Monocular blindness.** 102 girls, under 17 yrs. of age, seen at Undaan, 1/8'66-1/8'67. Visual acuity: **less than 1/10** in one eye, more in the other. Classification by site/type and etiology.

	12	13	18	20	21	28	40	67	89	91	98	99	Total
122										2			2
130							1					1	2
147									1				1
150							1	2				1	4
190												1	1
211	1												1
212	3												3
311											1		1
212					5								5
313	1			4		1	6					5	17
315	2		1					6			1	7	17
316	1			1									2
380.5	1	1						5				2	9
360								20					20
410							3		2				5
510											1		1
670										6			6
710												1	1
810												1	1
940										2		1	3
Total	9	1	1	5	5	1	11	33	3	10	3	20	103

Table A 51. **Monocular blindness.** 374 men, 16—41 yrs of age, seen at Undaan, 1/8'66—1/8'67. Visual acuity **less than 1/10** in one eye, more in the other. Classification by site/type and etiology.

	12	18	19	20	20.1	21	28	40	65	67	89	91	98	99	E	Ex	Re	Total
110												6						6
122												9						9
130								3					3	4				10
144											1							1
146											1							1
147											2							2
150	1			1				2						7				11
180								1										1
190								2						6				8
212	8																	8
311													3					3
313	2	2	2	16	1		1	22					15	63			1	125
315	7	2	2	1				3		2			2	33				52
316	5													2				7
318												1						1
320														1				1
380								2										2
380.4											1		1					2
380.5	1							1						11				13
390								1								1		2
410				1				17			3	29						50
510	1		5					1					16	5				28
530													1					1
550			1															1
560													1	1				2
610													1	1	1			3
640												1						1
660			1					2					2	2				7
710			3					1	1				1	1				7
810						1		2					1					4
940														1				1
990													1	3				4
Total	25	4	14	19	1	1	1	60	1	2	8	46	48	141	1	1	1	374

E : Eclips
Ex : Exophth.
Re : Recklinghausen.

Table A 52. **Monocular blindness.** 219 women, 16-41 years of age, Undaan, 1/8'66-1/8'67. Visual acuity **less than 1/10** in one eye, more in the other. Classification by site/type and etiology.

	12	18	19	20	40	66	67	89	91	98	99	Total
110									14			14
122									5			5
130					2					1	3	6
141								1				1
149								1				1
150	4		1	2			1				5	13
190	1										6	7
212	8											8
313			3	26	5					4	29	67
315	5	1		1						2	28	37
316	2									3	2	7
380	1											1
380.4				1	1							2
380.5										1	2	3
390				1								1
410					2			1	18			21
480									1			1
510			1							7	1	9
530										1		1
610					1					1		2
640									1	1		2
660										1		1
710										1	2	3
810					1	1						2
940											1	1
990											3	3
Total	21	1	5	31	12	1	1	3	39	23	82	210

Table A 53. **Monocular blindness.** 341 men, over 40 yrs. of age, Undaan 1/8'66-1/8'67. Visual acuity **less than 1/10** in one eye, more in the other, Classification by site/type and etiology,

	12	18	19	20	21	28	40	46	62	89	91	98	99	Ca	Total
110											17				17
122											2				2
130							1						4		5
142										1					1
150	2			2			1	1					6		12
212	2														2
311												1			1
313			5	11		4	15					6	44		85
315		1		1		1	1					2	11		17
316													2		2
280.4				2									5		7
380.5													1		1
410				4			4		3		122				133
510			3		2						1	11	2		19
530			1									1			2
550												1			1
560												2			2
610			1		1							1			3
640													1		1
650										1					1
660												2			2
710			5		1							5		1	12
810							1					4	2		7
940												1			1
990												1	4		5
Total	4	1	15	20	4	5	23	1	3	2	142	38	82	1	341

Ca : Carcinoma.

Table A 54. **Monocular blindness.** 259 women, over 40 yrs. of age, Undaan, 1/8'66-1/8'67. Visual acuity **less than 1/10** in one eye, more on the other. Classification by site/type and etiology.

	12	19	20	40	62	64	91	98	99	Ca	Total
110							39				39
122			1				1				2
130									1		1
150				2					4		6
190									1		1
212	1										1
313			15	1				1	21	1	39
315	1		3	1					10		15
316								1	2		3
318				1					1		2
380.4			1						1		2
380.5									2		2
390								1			1
410			2	1	2		101				106
480							1				1
510		2						9	1		12
530								1			1
560								2			2
570				1							1
610					1	3		2	1		7
640									3		3
660								1			1
710		3						1	1		5
810		1						1	1		3
990								1	2		3
Total	2	6	22	7	3	3	142	21	52	1	259

Ca : Carcinoma.

Table C 23. **Monocular blindness.** Major causes of blindness in age groups by sex. Undaan, 1/8'66—1/8'67. Visual acuity less than 1/10 in one eye, more in the other. Compare Tables A 49—54 and Table, 9, Sorsby, 1966, p. 13.

Cause	0—17 yrs				16—41 yrs				Over 40 yrs.			
	M		F		M		F		M		F	
Congenital malformations	4	3.4%	3	3%	8	2.1%	3	1.3%	1	0.3%	—	—
(Congen. catar.)	(3)		(2)		(3)		(1)					
Retinoblastoma	6	5.1%	6	6%	—	—	—	—	—	—	—	—
Ophth. neonatorum	—	—	1	1%	—	—	—	—	—	—	—	—
Conj. gonorrh. non-neonatorum	5	4.2%	8	8%	25	6.6%	21	9.5%	4	1.2%	2	0.7%
Keratomalacia	31	26.5%	33	33%	2	0.5%	1	0.4%	—	—	—	—
Retinit. pigment.	—	—	—	—	—	—	—	—	1	0.3%	—	—
Corneal disease, Wr +	1	0.8%	—	—	4	1.7%	3	1.4%	5	1.4%	—	—
Idem, other	7	6%	24	24%	139	37%	72	33%	76	22%	41	16%
Uveitis, Wr +	—	—	—	—	6	1.6%	1	0.4%	4	1.2%	2	0.7%
Idem, other	3	2.5%	1	1%	24	6.4%	9	4.1%	20	5.8%	13	5%
Ophth. sympathica	—	—	—	—	—	—	—	—	—	—	1	0.4%
Glaucoma	1	0.8%	—	—	6	1.6%	14	6.3%	17	5%	39	15%
Senile cataract	2	1.7%	—	—	29	7.7%	18	8.2%	125	36.6%	103	40%
Trachoma	3	2.5%	5	5%	20	5.3%	31	14%	20	5.8%	22	8.4%
Trauma	30	25.6%	11	11%	60	16%	12	5.4%	24	7%	7	2.7%
Retinal detachment	—	—	—	—	1	0.26%	2	0.8%	1	0.3%	3	1.1%
Optic atrophy, luetic	—	—	—	—	3	0.8%	—	—	5	1.4%	3	1.1%
Idem, other	1	0.8%	1	1%	3	0.8%	3	1.4%	7	2%	2	0.7%
All other causes	23	20%	9	9%	44	11.7%	29	13.2%	31	9%	21	8%
Total	117	100%	102	100%	374	100%	219	100%	341	100%	259	100%

Table C 24. **Monocular blindness.** Causes of blindness in age groups by sex. Undaan, 1/8'66-1/8'67. Visual acuity **less than 1/10** in one eye, more in the other. Adjusted female incidence against actual male incidence.

Cause	0-17 yrs		16- 41 yrs		Over 40 yrs	
	M	F	M	F	M	F
Congenital malformations	4	3	5	1	1	—
(congen. catar.)	(3)	(2)	(3)	(<1)	—	—
Retinoblastoma	6	5	—	—	—	—
Ophth. neonatorum	—	<1	—	—	—	—
Conj. gonorrh. non-neonat.	5	7	25	17	4	2
Keratomalacia	31	28	2	1	—	—
Retinitis pigmentosa	—	—	—	—	1	—
Corneal disease, Wr +	1	—	4	2	5	—
Idem, other	7	20	139	57	76	38
Uveitis, Wr +	—	—	6	<1	4	2
Idem, other	3	<1	24	7	20	12
Ophthalmia sympathica	—	—	—	—	—	<1
Glaucoma	1	—	6	11	17	36
Senile cataract	2	—	29	14	125	95
Trachoma	3	4	20	24	20	12
Trauma	30	9	60	9	24	6
Retinal detachment	—	—	1	2	1	3
Optic atrophy, luetic	—	—	3	—	5	3
Idem, other	1	<1	3	2	7	2
All other causes	23	8	44	23	31	19
Total	117	(87)	374	(173)	341	(221)

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Abbreviations : AJO : American Journal of Ophthalmology.
GTvNI : Geneeskundig Tydscrift voor Nederlandsch-Indie.
JAMA : Journal of the American Medical Association.
KMA : Klinsche Monatsblätter für Augenheilkunde.
NTvG : Nederlandsch Tydschrift voor Geneeskunde.

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STATEMENTS

I. High priority should be given to measures for preventing Xerophthalmia, which is the most frequent cause of blindness in young children in many parts of Indonesia. Either the general use of Red Palm Oil in daily oral doses for small children could be popularized by all useful means, or a system could be developed, by which all children up to age 4 years would receive injections of 200.000 IU Vitamin A semi-annually, or both methods could be adopted.

II. As Glaucoma as a potentially blinding disease in persons over 40 years of age often is discovered too late, it would be advisable to urge the public to have an eye examination by an ophthalmologist for prescription of eye glasses at least once in ten years over the age of 40.

III. It seems that Trachoma in its severe forms is less frequently seen now than in former decades ; in devising campaigns for its eradication this should be taken into account.

IV. At least one wave of higher incidence of congenital cataract has been observed in Surabaja. Generally Rubella in the mother is recognized as the most probable cause of cataract and other congenital malformations in the child. Therefore it would be very desirable to apply modern methods of serology and immunology to determine whether Rubella is encountered in Indonesia, and after that whether this would necessitate measures in the case of mothers exposed to Rubella in the first trimester of pregnancy.

V. For the study of the epidemiology of Sclerosis multiplex it would be useful to investigate the incidence of this disease among the large group of persons of Indonesian stock and their families, who settled in the Netherlands around 1950, as the disease is very rare in Indonesia and far more frequent in the Netherlands.

VI. As sulfa-drugs can cause various dangerous side-effects, e.g. Leukopenia, anuria, & an exanthem resembling Stevens-Johnson's syndrome, the indiscriminate use of these drugs should be discouraged.

VII. In an era when traffic accidents are on the increase, the knowledge of the signs and symptoms of blow-out fracture of the orbit becomes important.

VIII. Indications that badly-regulated diabetes, especially in young individuals, leads to earlier and more dangerous complications than well-regulated diabetes, necessitate a warning to diabetics, that only regular check-ups on their disease can prevent dangerous developments as far as possible.

IX. If it is possible to collect sufficienly large statistical figures on certain diseases in a centrally located hospital, these figures need not be of less value than those collected in random field surveys, also because the examination of the individual cases can be more complete.

X. Rehabilitation of various kinds of handicapped children and adults, should primarily be started in close cooperation with medical specialists in the field, so as to reach the best result for the patient.

XI. The use of a white cane for walking by the blind and the deaf, would enhance their freedom of movement and their safety outside their homes. In popularizing its use the general public should be urged to pay attention to people who walk with a white cane.

XII. As diseases in tropical climates and those in colder regions tend to differ, and as increasing international traffic increases the chances of finding diseases outside their usual habitat, it would be useful if it were possible by exchange programs for non-tropical doctors to work for a period of circa one year in the tropics, and for tropical doctors to work in colder climates.

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